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THE CHIASMAL SYNDROME

OF PRIMARY OPTIC ATROPHY AND BITEMPORAL FIELD DEFECTS IN ADULTS WITH A NORMAL SELLA TURCICA *

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Though the ophthalmic surgeon, since the days when George Bartisch of Dresden wrote his celebrated "Augendienst," has almost wholly restricted his operative procedures to lesions within the orbital boundaries, he nevertheless has been obliged to look beyond the orbit into the territory of the neurologist for an explanation of many of the causes of disturbed vision. Helmholtz' discovery of the ophthalmoscope first made this possible, as von Graefe and Donders were quick to appreciate; and ere long the neurologists under the insistence of Hughlings Jackson and Gowers found that the routine examination of the fundus oculi was no less important to them than to the ophthalmologist. Thus the general employment of the ophthalmoscope for diagnostic purposes and the subsequent gradual development of perimetry by von Graefe, Förster, Bjerrum, Rönne and others served to emphasize the overlap between ophthalmology and neurology which had its first notable expression in the classic monograph of Wilbrand and Saenger published twenty years ago.

A somewhat academic interest in the diagnosis and treatment of lesions affecting the optic chiasm might well enough have remained solely in the hands of ophthalmologists and neurologists had it not been for the rapid growth in the past two decades of our knowledge of pituitary disorders, the clinical recognition of which depends in large measure on the concomitant deformations of the sella turcica. Consequently, through Roentgen's discovery, no less than through the invention of ophthalmoscopy and perimetry, is the present discussion made possible, for without the x-ray we would scarcely be considering the chiasmal syndrome due to suprasellar tumefactions as something apart from the syndrome of tumors that arise within and expand the sella turcica.

So in their turn the roentgenologist and the endocrinologist have come to share the interest of the ophthalmologist and neurologist in this

* An address before the International Ophthalmological Congress, Scheveningen, Sept. 12, 1929. Since its publication in the Transactions of the Congress, this paper has undergone revision, its data have been brought up to date, and an example of a chiasmal glioma in an adult has been added.

highly important region. But present-day neurologists—or many of them—in imitation of the ophthalmologist are becoming surgicalized, and though neurologic surgery covers a large field, its more serious problems are those produced by intracranial lesions which in one way or another implicate vision. In general terms, therefore, the modern neurosurgeon has come to deal largely with lesions that mechanically affect the central apparatus of vision while the ophthalmic surgeon limits his operative procedures to the intra-orbital portion of this apparatus. One in a sense is an extracranial, the other an intracranial, ophthalmic surgeon, neither of them venturing to trespass much beyond the narrows of the optic foramina.

Through the perfection of neurosurgical technic, the heretofore largely inaccessible region of the chiasm has come of late years to be one frequently and readily exposed to view, and in consequence one is better prepared to discuss the nature of the many disorders that produce *the syndrome of primary atrophy and bitemporal hemianopsia* (or a tendency in that direction) *when the sella turcica remains* (comparatively speaking) *unaffected.*

Attention has elsewhere been drawn [1] to this particular syndrome as it occurs in middle-aged patients in association with tumors of one particular type: namely, the suprasellar meningiomas that arise from the sulcus chiasmatis and tuberculum sellae. Stress was laid in this earlier paper on these particular lesions for the reason that of all the suprasellar tumors that implicate the chiasm they are the most favorable for operation. This being so, it is incumbent on ophthalmologists, who are likely to be primarily consulted, to recognize them at the earliest possible stage.

The advantage to a surgeon of knowing with reasonable certainty before opening the skull not only the situation of the lesion he is to attack but its precise histologic nature has been frequently emphasized; and one might suppose, in view of the experience already gained with the syndrome in question, that fewer diagnostic mistakes would occur. As a matter of fact, in the nine months since the last case included in our previous report, the provisional preoperative diagnosis of suprasellar meningioma has been made eleven times, with eight diagnostic errors: two of the patients had a Rathke's pouch cyst without calcification, another an aneurysm, four others chromophobe adenomas and still another a local chronic arachnoiditis. It becomes evident, therefore, that in the present state of our knowledge a differential preoperative pathologic diagnosis is difficult; and it is perhaps natural enough that in the case of adults showing a suprasellar syndrome a meningioma should have been the diagnosis most often favored.

1. Cushing and Eisenhardt: Meningiomas Arising from the Tuberculum Sellae, Arch. Ophth. **1**:1-41 and 168-205 (Jan. & Feb.) 1929.

In view of my personal interest in the subject of the intracranial meningiomas in general, I may be permitted to use the chiasmal syndrome of these tumors as the primary basis of comparison with the scarcely distinguishable syndromes produced by tumors of other sorts in the same suprasellar situation.

THE SUPRASELLAR MENINGIOMAS

These tumors, as previously pointed out, have their point of dural attachment over the chiasmatic sulcus and tuberculum sellae so that when freed at this growing point, the major unattached portion of the

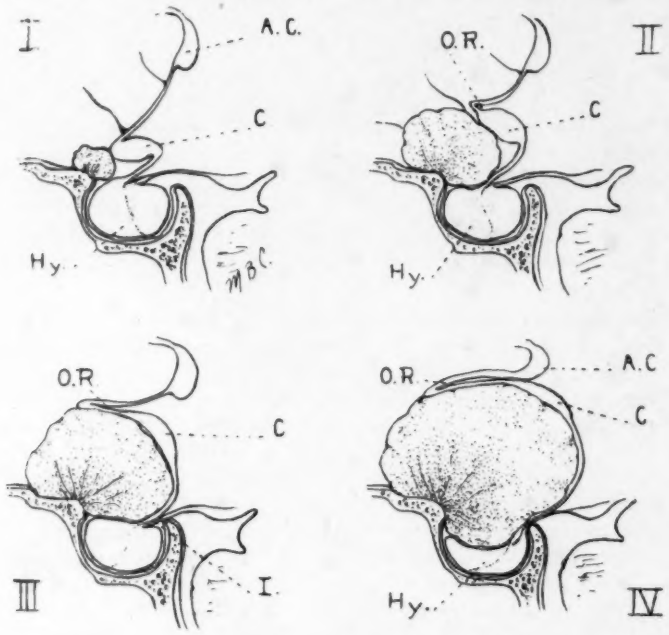


Fig. 1.—To illustrate on sagittal section four stages of the advancing deformation of chiasm and third ventricle produced by a meningioma.

growth which underlies and elevates the chiasm can usually be tilted out in an intact piece.

The relation of tumors of varying size to the degree of chiasmal deformation was represented schematically in the accompanying sketch (fig. 1) borrowed from our earlier paper. By the time the growth has attained dimensions much in excess of the largest of the tumors there diagrammatically shown, not only will its surgical dislodgment become difficult or impossible, but the simple syndrome under discussion will have become more or less obscured by the effects of hydrocephalus, by involvement of the hypothalamic centers, by evidences of pituitary insufficiency and by pressure absorption of the sella.

Since the original report of fifteen cases, two additional patients have been operated on, and these cases may deserve recording here in full.[2] In the first of them the crossed bundles must have been unduly stretched or traumatized in the process of dislodging the growth for the temporal fields have never completely filled out.

CASE 1.—*Progressive failure of vision for twenty-eight months. Bitemporal though unequal field defects and questionable optic atrophy in the absence of other symptoms in a middle-aged woman with normal sella. Clinical diagnosis of suprasellar meningioma. Operation with enucleation of a tumor weighing about 10 Gm. Recovery with ultimate improvement in vision.*

Aug. 23, 1928: Admission of Mrs. E. L. W., 48 years of age, on the recommendation of Dr. Gertrude Johnson of Battle Creek, Mich., with the diagnosis of pituitary tumor.

History.—The patient, a bank teller by occupation, in *April, 1926,* first became aware of some obscuration of vision in the left eye. She was fitted for glasses without benefit. In *October, 1926,* fields of vision were first taken and showed a marked left temporal defect with a central scotoma. Vision in this eye became progressively worse, and ere long the right eye began to be similarly involved, so that in *June, 1928,* she was obliged to relinquish her occupation. She had been seen by a number of competent ophthalmologists, who agreed that there was no change in the eyegrounds that could explain the loss of vision. A tonsillectomy was performed and teeth were extracted without favorable effect.

Physical Examination.—Apart from the visual defects to be described, the examination was wholly negative. The patient, an intelligent and cooperative woman, was in excellent general health and free from physical discomforts of any kind. There were no evidences of pituitary insufficiency; the basal metabolic rate of —15 per cent could not be so interpreted, since the rate rose to +14 per cent after partaking of food. Though 48 years of age, her menstrual periods continued regular and normal. Her weight had not increased, nor had she ever observed polydipsia or polyuria.

The sella turcica (fig. 2) was regarded as normal, though attention was drawn to the slight apparent deformation of the posterior clinoid processes. Stereoscopically, a slight roughening and increased density of the tuberculum sellae were observed by the roentgenologist (M. C. Sosman).

Ophthalmologic Signs.—The fields of vision showed unequal though unmistakable bitemporal field defects, the boundaries of which were somewhat variable from day to day, the widest peripheries being shown in the chart (fig. 3). Visual acuity on the left, reduced to counting fingers with difficulty, was

2. A third additional case (surgical no. 25758) was that of a woman aged 51, whose symptoms of seven years' duration had progressed beyond the simple chiasmal syndrome in question. The hazards of intervention were made clear and she sought advice in another city, where the tumor, which proved to be a meningioma, was attacked with postoperative fatality. In a fourth case (surgical no. 34443), too late to include in the original report, the patient was successfully operated on July 24, 1929. In still another case (surgical no. 29902), with correct clinical diagnosis, the patient refused operation and recently died, blind, two years after she was seen, a postmortem examination verifying the diagnosis. All told, therefore, there have been twenty verified cases of suprasellar meningiomas in the hospital records. Eighteen of these patients have been operated on, with a single death from an avoidable accident.

Fig. 2 (case 1).—Profile view of approximately normal sella of patient with suprasellar meningioma.

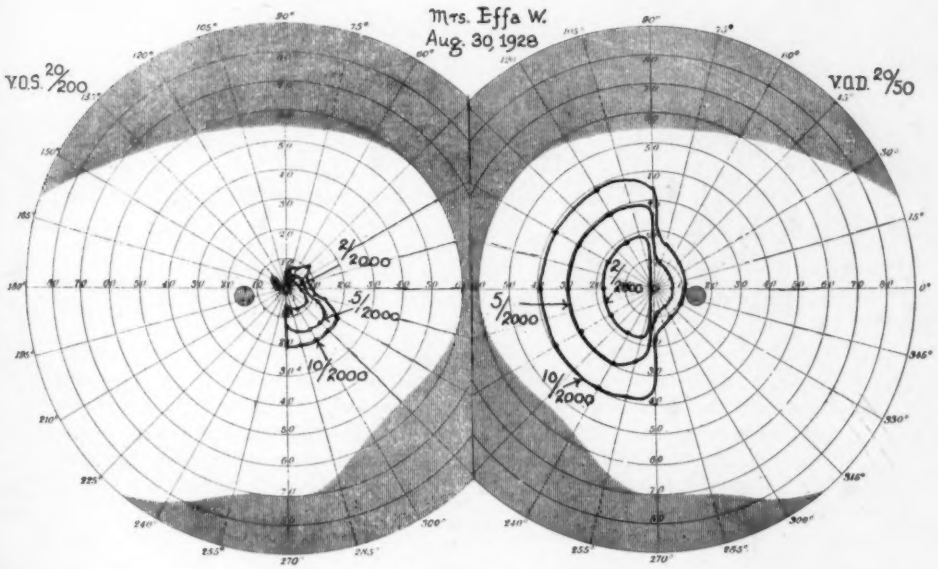


Fig. 3 (case 1).—Preoperative fields of vision (screen method only), Aug. 30, 1928.

estimated on her better days at 20/200; on the right, it was 20/50. In view of the marked field defects, the fundi showed surprisingly little change and had been regarded as normal by our predecessors (Dr. L. V. Stegman and Dr. W. R. Parker). At the time of admission, however, the left nerve when compared with the right showed, as we thought, an unmistakable slight pallor, an opinion which was corroborated by Prof. G. S. Derby, who kindly examined the patient's eyegrounds to check this particular point.

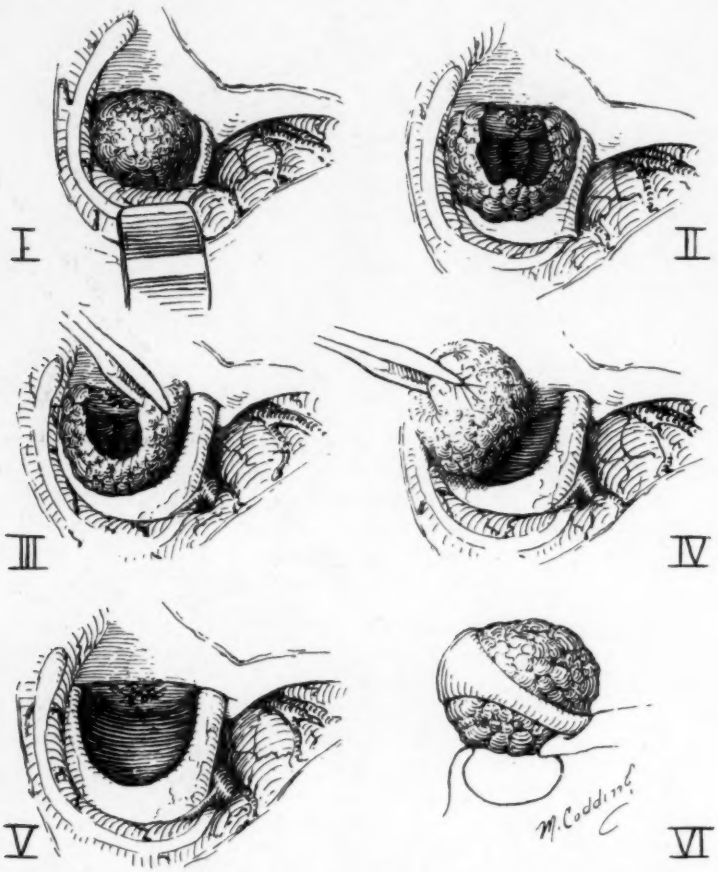


Fig. 4 (case 1).—Elaboration of immediate postoperative sketches indicating the stages of the surgical procedure. *I*, the primary view of the partly concealed right optic nerve with finely nodular tumor beyond. *II*, the primary electrosurgical "scalloping" of the lesion to free its dural attachment over the sulcus chiasmatis. *III*, *IV* and *V*, the tilting out of the posterior portion of the growth, leaving a smooth-walled excavation. *VI*, estimated size and relation of growth to the sella and chiasmal ring.

The patient was discharged on *August 31*, and reentered the hospital for further study three weeks later. There had been no discernible advance in the process during this interval.

Operation.—On *Sept. 21, 1928,* under procaine-ether anesthesia, the customary right transfrontal osteoplastic flap was reflected with some difficulty, owing to a densely adherent dura. On exposing the right optic nerve, the typical, finely nodular surface of a suprasellar meningioma was exposed. By the method shown in the sketches (fig. 4), the growth was first freed from its attachment to the tuberculum by electrosurgical methods, and it was then possible without great difficulty to dislodge from under the chiasm the intact posterior portion (fig 5). The dura over the tuberculum was thoroughly heated with the coagulating current. After the usual painstaking hemostasis, the flap was replaced and the wound closed.

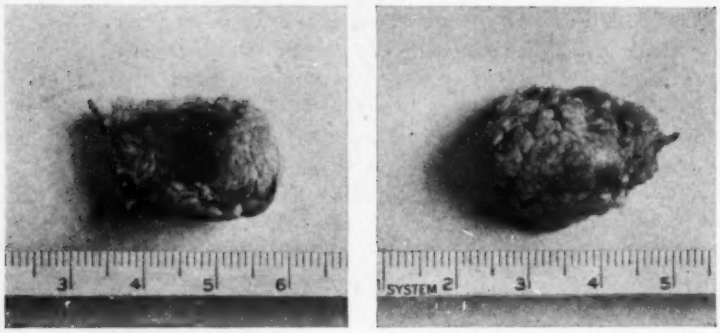
Postoperative Notes.—The patient made an excellent recovery from the operation, but as she showed a less rapid return of vision than was expected, the formation of a postoperative clot was feared. On the third day, therefore, under local anesthesia the flap was reelevated without disclosing the expected hematoma. Not until two weeks had passed did she begin to appreciate any subjective improvement in vision. Even before this time, however, it was evident from perimetric tests that the greatly constricted nasal field on the left had begun to widen.

Wound healing was excellent (fig. 6), and our last fields, taken on *October 19,* a week after the patient's discharge, showed (fig. 7) fairly symmetrical bitemporal hemianopsia with sufficient gain in the left eye, where complete and full restoration of the nasal field had occurred, to compensate for the slightly increased temporal constriction in the right macular area. The acuity on the left had improved from 20/200 to 20/100 and on the right from 20/50 to 20/20.

The tumor proved histologically to be a typical fibroblastic meningioma (fig. 8).

Subsequent Notes.—Frequent reports from Dr. Johnson as well as from the patient told of slow though steady improvement with some slight return of vision in the upper temporal fields after six months (fig. 9): vision in the left eye was 20/80 + 2 and in the right eye 20/15 + 1. The patient states that she is able to read the finest print and thread the finest needle without difficulty.

Comment.—In this case the field defects were wholly disproportionate to the ophthalmoscopic findings, for even the eye chiefly involved showed after two years of advancing blindness so slight a degree of pallor that it would easily have passed as normal. The operation, so far as the enucleation of the tumor was concerned, was not difficult, and there was every reason to expect complete restoration of the defective bitemporal vision. That this did not occur may have been due either to some unobserved traumatism or to undue stretching of the chiasm in dislodging the growth from its embrace, or possibly to overenergetic electric coagulation of the dura over the tuberculum which may have damaged the crossed nerve bundles of the adjacent nerves.



a *b*

Fig. 5 (case 1).—Posterior tumor fragment: (*a*) the anterior, partly excavated surface; (*b*) the posterior surface of the finely nodular lesion.

Fig. 6 (case 1).—Two weeks after operation, to show site and condition of the twice opened wound.

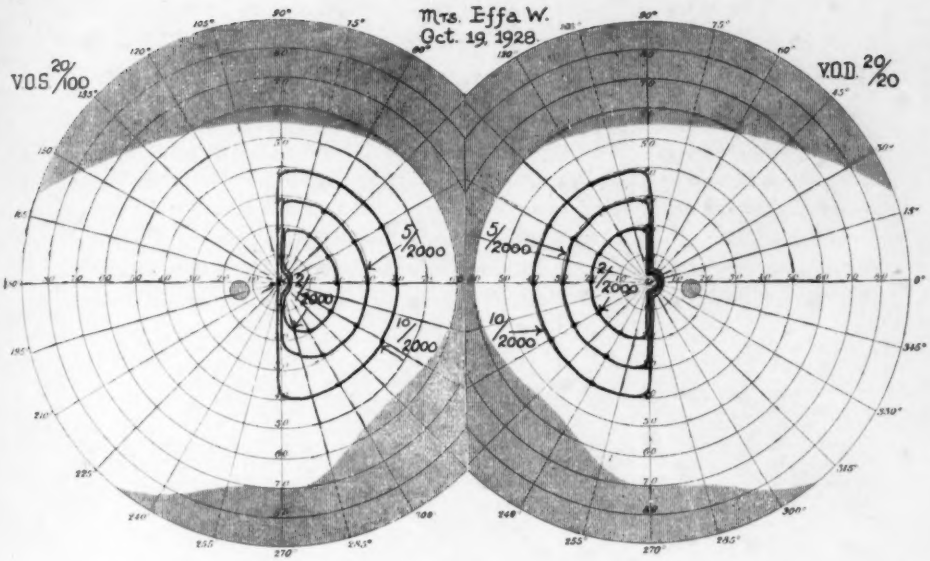


Fig. 7 (case 1).—Postoperative fields (screen method) for comparison with figure 3.

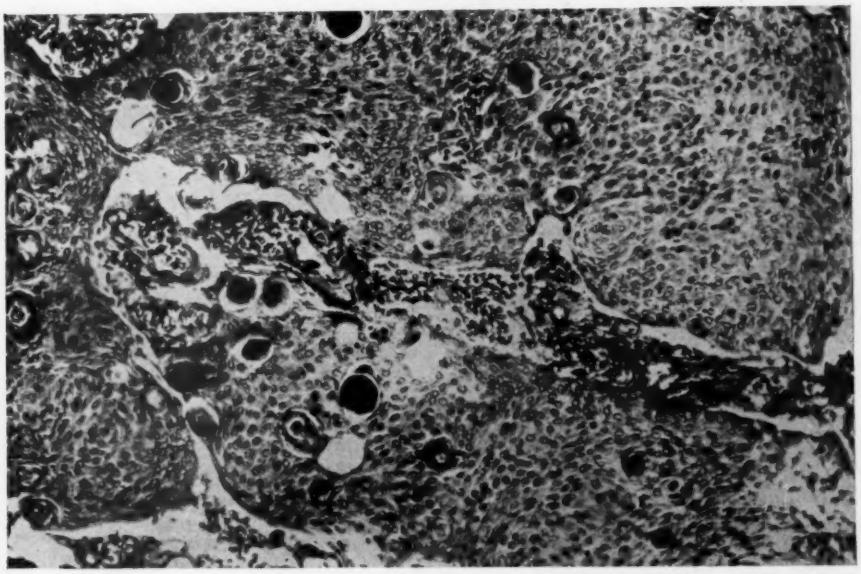


Fig. 8 (case 1).—To show the fibroblastic, slightly psammomatous meningioma. Phosphotungstic acid-hematoxylin; × 150.

A better result was obtained in the following case in spite of the fact that the atrophy was more pronounced, the symptoms therefore being more typical of the syndrome under discussion.

CASE 2.—*A middle-aged woman with progressive failure of vision for two years. Typical bitemporal hemianopsia with primary optic atrophy and an essentially normal sella. Clinical diagnosis of suprasellar meningioma corroborated at operation. Enucleation of tumor with prompt restoration of normal vision.*

Dec. 6, 1928: Admission of Mrs. Jennie P., a Polish housewife, 40 years of age, on the recommendation of Dr. Joseph J. Skirball of Boston, with the complaint of failing vision.

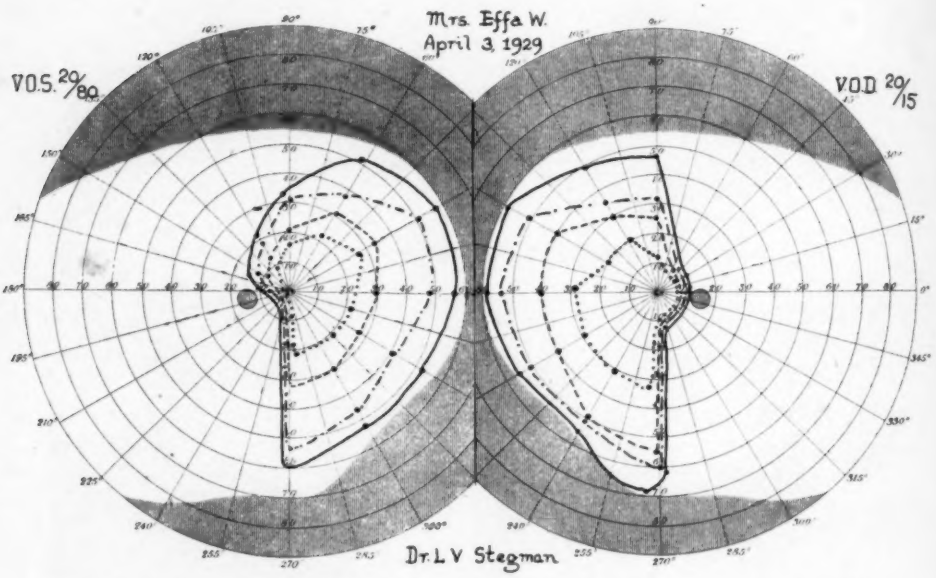


Fig. 9 (case 1).—Fields to perimeter for form and color six months after operation (kindness of Dr. Stegman).

History.—The patient, the mother of two healthy children, had always been vigorous and well. Her catamenia had showed no irregularity; her weight of about 82 Kg. had long been stationary.

In 1926 she first observed a blurring of vision in the left eye and a year later in the right eye. There were no other symptoms. She consulted Dr. Skirball in *June, 1928,* at which time he found no abnormalities apart from a marked myopia easily corrected by glasses and a slight shrinkage of the left temporal field of vision.

Physical Examination.—The patient's sole complaint was loss of vision. The results of the examination were wholly negative apart from hypertrophic tonsils, a basal metabolic rate of —17 per cent and the following ophthalmologic findings.

The fields of vision (fig. 10) showed a typical complete bitemporal hemianopsia. Vision in the right eye was 20/70, and in the left eye owing to total loss of macular vision, it was estimated at less than 5/200, the ability to count fingers being retained in the nasal field. Both nerves, particularly the left, showed sharp outlines and had a distinctly atrophic pallor. The sella (fig. 11) was regarded as within normal limits, though the posterior clinoid was somewhat thinned and possibly pushed backward.

The clinical diagnosis favored a suprasellar meningioma though a pituitary adenoma was regarded as possible.

Operation.—On *Dec. 20, 1928,* wholly under procaine anesthesia, the customary right frontal osteoplastic flap was reflected without difficulty, and on

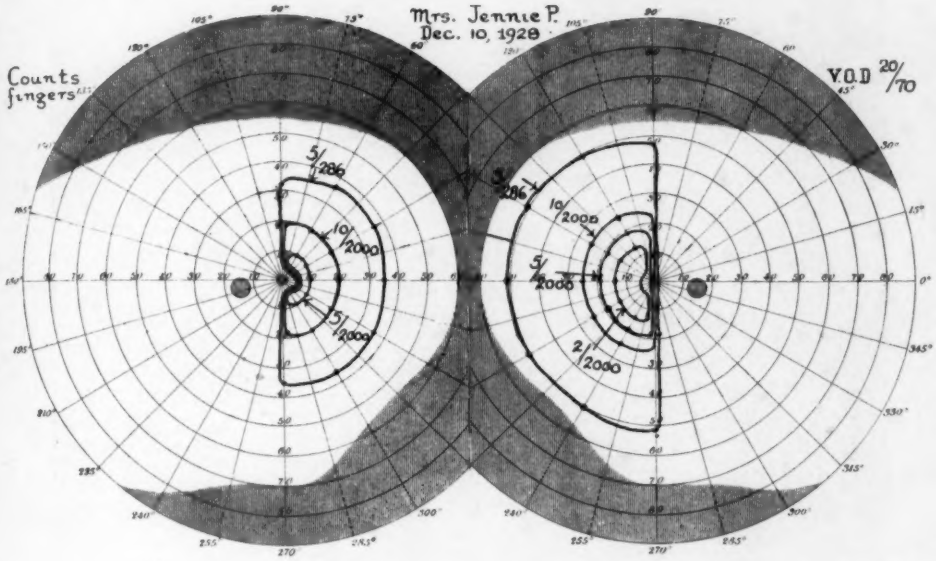


Fig. 10 (case 2).—The fields of vision to perimeter and screen on the patient's admission.

elevating the frontal lobe the usual dural incision was made along the sphenoidal ridge. A large amount of cerebrospinal fluid escaped, and the right optic nerve and adjacent tumor were easily exposed. The tumor, unlike most suprasellar meningiomas, had a perfectly smooth rather than a coarsely granular surface. Consequently the growth was at first mistaken for a suprasellar adenoma, and it was not until a fragment of tissue had been submitted for microscopic examination in the fresh that its true nature was certified.

The tumor, which was somewhat larger than in the preceding case, was treated in the usual fashion by primary electrosurgical excavation until freed from its dural attachment. It was then possible in the manner shown in the operative sketches (fig. 12) gently to tilt out the bulk of the growth bloodlessly

from its bed. Not until this enucleation was accomplished did the left optic nerve come into view on the distant side of the surprisingly large and deep residual pocket. Beneath the nerve the left carotid artery was clearly exposed. In the posterior floor of this pocket could be seen the flattened infundibulum which evidently contained fluid, as could be told by compressing it and then allowing it to refill. After careful hemostasis, the flap was replaced and the scalp closed in the usual detail without drainage.

The tumor, the major fragments of which are shown (fig. 13), was estimated to weigh about 14 Gm. Its histologic architecture is shown in figure 14.

Postoperative Notes.—At the conclusion of the operation, which was exceptionally well borne, the patient was immediately conscious of improvement in

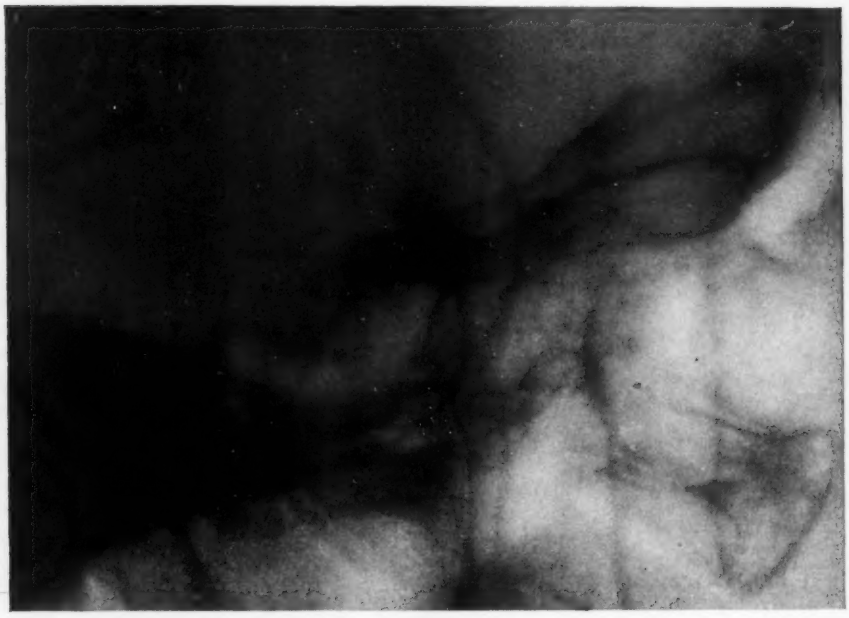


Fig. 11 (case 2).—Showing profile of essentially normal sella.

vision. On the following day when the dressing was changed and the sutures removed, a return of sight in the previously blind fields was easily demonstrated by rough tests. She was sitting up on the fourth day *(December 24),* when the fields were first taken on the screen. They showed practically normal temporal peripheries in the left (the previously worse) eye, whereas the right (the better eye) was lagging behind. The acuity was not measured, but there was marked subjective improvement. Three days later *(December 27)* fields, taken to both perimeter and screen, showed further improvement (fig. 15) with a normal acuity of 20/20 in each eye and only a slight residual constriction of the upper temporal fields in the right eye. The patient's surgical recovery was uneventful, with perfect wound healing (fig. 16), and she was discharged on *Jan. 8, 1929.*

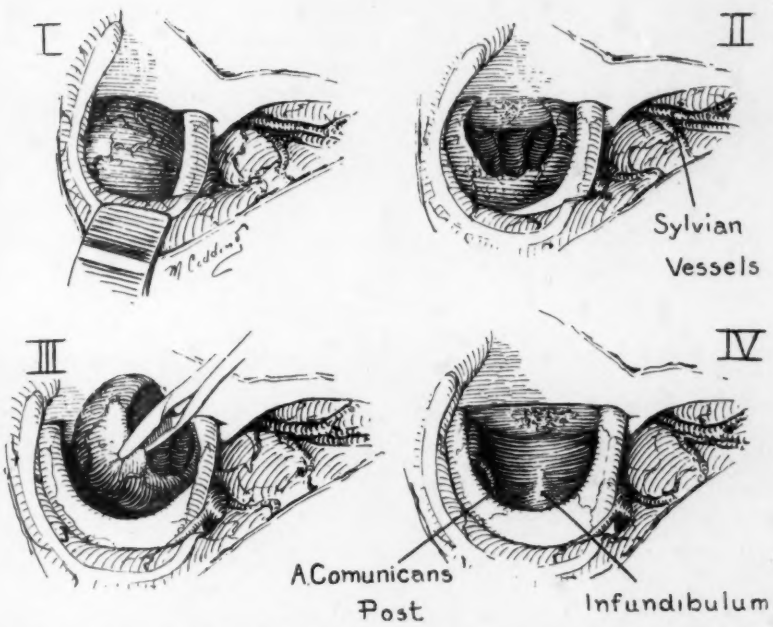


Fig. 12 (case 2).—Operative sketches, showing: *I*, the smooth-surfaced lesion suggesting an adenoma when first exposed; *II*, primary electric scalloping of the growth; *III*, the posterior part of tumor being tilted out; *IV*, an oblique view into the bed of the tumor showing the left carotid and the infundibular stalk entering the pituitary diaphragm.

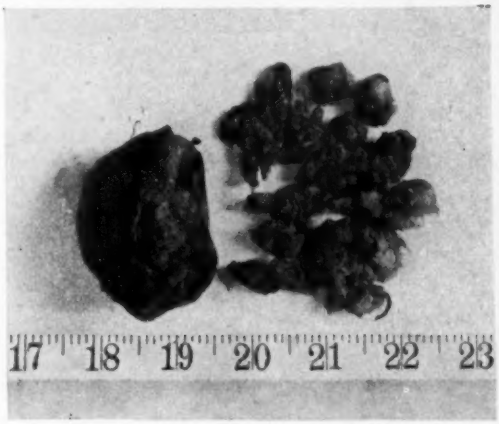


Fig. 13 (case 2).—Showing the intact posterior portion of the tumor with some of the electrically removed fragments primarily excavated from it.

Subsequent Notes.—The patient reported for examination six weeks after her discharge, at which time a possible slight residual pallor of the left optic nerve was apparent. A letter from Dr. Skirball of *May 8, 1929,* stated that he was able to find no trace of any former trouble in the fundi, fields or visual acuity.

Comment.—The restoration of normal vision in this case was unusually rapid and complete. The case shows what may be expected of these operations in the future when the syndrome has become sufficiently well known for the lesion to be exposed at a still more favorable stage.

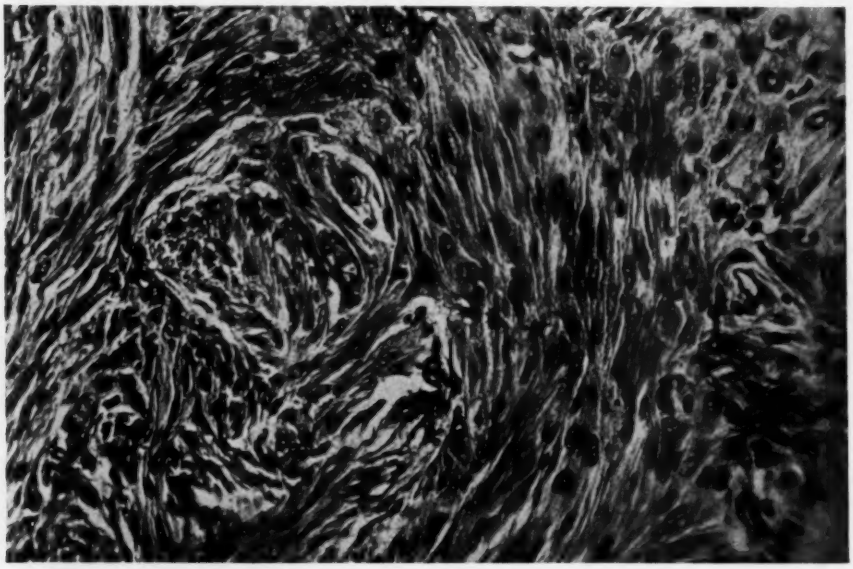


Fig. 14 (case 2).—Photomicrograph of the fibroblastic meningioma. Phosphotungstic acid-hematoxylin; × 300.

The question of the proper treatment of the infiltrated dura after the enucleation of one of these tumors has given no little concern. That nests of viable cells in the interstices of that portion of dura to which the growth is attached are a potential source of recurrence unless the membrane and tumor are removed together, is true of all meningiomas wherever they arise. This joint removal is easily accomplished in the accessible meningiomas of spinal canal or cerebral vault, but in the case of the tumors that arise from the cranial base, from which the dura strips with difficulty, recourse must be had to the electrosurgical coagulation of the viable cells in the adherent membrane. To be sure, there is probably less risk of recurrence after the removal of one of

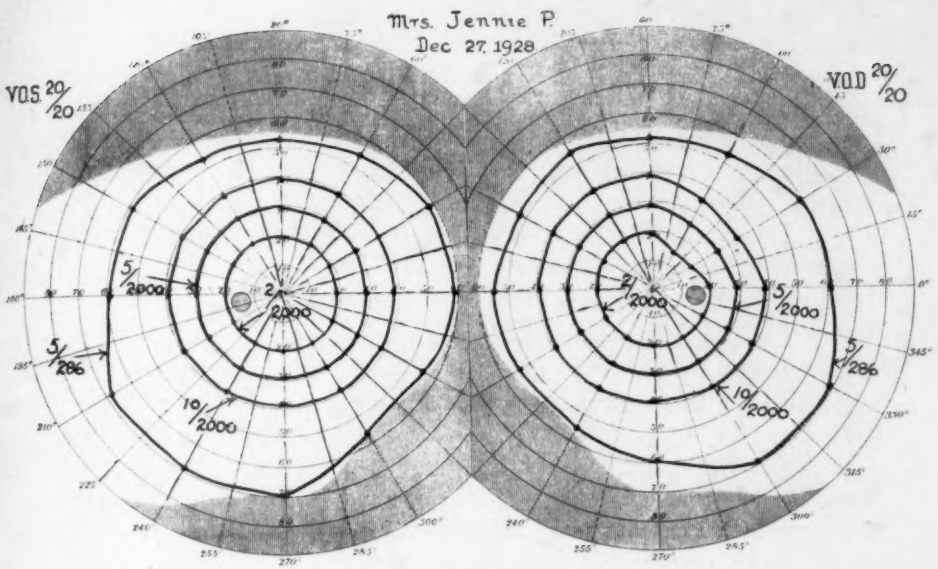


Fig. 15 (case 2).—Fields to screen and perimeter seven days after operation (cf. fig. 10).

Fig. 16 (case 2).—The patient before discharge, to show the nigh invisible condition of the wound. Before the operation she had been given therapeutic radiation on the mistaken assumption that she had a pituitary adenoma.

these somewhat fibroblastic meningiomas than there is with the more highly cellular and actively growing types of meningiomas that are met with in other regions. This, however, is a surgical rather than an ophthalmologic problem, and it may suffice to say that in none of the suprasellar meningiomas in the series (the oldest of them now thirteen years after the operation) has there been as yet any symptomatic evidence of recurrence even though in the earlier cases there was no effort, electrically or otherwise, to destroy the cell nests in the area of dural attachment.

THE SUPRASELLAR ADENOMAS

Of all the lesions that produce bitemporal field defects with primary optic atrophy in adults or middle-aged persons, the adenomas of the hypophysial anterior lobe are the most common. This is a matter of general knowledge, and in the vast majority of cases the constitutional evidences of disordered pituitary function serve to make the seat and nature of the trouble unquestionable. Though this statement is perhaps less true of the chromophobe adenomas associated with hypopituitarism than with the chromophilic adenomas of acromegaly (hyperpituitarism), in either case the roentgenologic demonstration of a widely distended sella turcica leaves no doubt as to the cause of the disturbed vision.[3]

Enlargement of the pituitary fossa, however, does not invariably accompany a pituitary adenoma. The lesion may conceivably arise from an "anlage" in the hypophysial stalk above the membranous diaphragm of the pituitary capsule, or if superficially placed it may escape through the diaphragm without having previously expanded the sella.[4] Under these circumstances, the remainder of the gland being unaffected by

3. As a matter of fact, the chromophobe adenomas that affect vision are far more common, there having been 219 verified examples in our series to May 1, 1929, as opposed to 89 chromophile adenomas (including all cases of outspoken as well as of fugitive acromegaly). Acromegaly, in short, is a far less frequent disorder than the reverse state of hypopituitarism. What is more, the adenoma of acromegaly in the long run is less actively growing than the chromophobe adenomas and consequently less likely to attain a size sufficient to involve the chiasm. It may be noted also that chromophobe adenomas may occasionally occur in childhood; the youngest case in our series was that of a child 10 years of age.

4. Just how this early escape of an adenoma from the sellar confines may come about is not wholly clear, but it probably has something to do with the imperfect formation of the dural diaphragm which ordinarily serves so to confine an adenoma within the pituitary fossa that its bony walls will expand widely from pressure long before the dense diaphragm bulges upward sufficiently to compress the optic nerves and chiasm. It is of course well known that even a completely formed diaphragm may ultimately give way and permit the tumor to grow freely in the intracranial chamber. The intracranial extrusion of the adenoma usually occurs through the opening for the pituitary stalk, but this is not an invariable rule.

pressure, the constitutional evidences of disordered hypophysial function are inconspicuous or absent, a disturbance of vision being the inaugural symptom.

The opportunity to investigate after death a chromophobe adenoma that has behaved in this way could scarcely be expected. An example, however, of a large extrasellar chromophile adenoma in an acromegalic patient with a comparatively unaffected sella and unimpaired vision has been described elsewhere.[5] In this case the adenoma, which attained a huge size in the temporal lobe, had burst through the dural capsule to one side missing the chiasm altogether, and though the residual of the gland that occupied the sella was somewhat compressed,

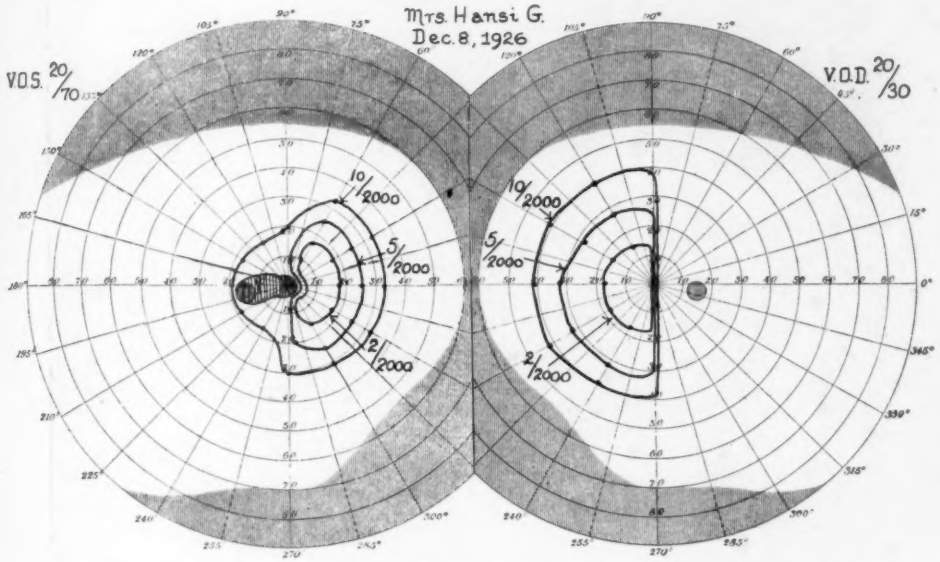


Fig. 17 (case 3).—Fields on admission of patient with chiasmal syndrome due to small suprasellar adenoma (screen method only with 10, 5 and 2 mm. disks).

it was otherwise normal. Though no comparable instance of an extrasellar chromophobe adenoma associated with an unexpanded sella has been encountered post mortem, a few examples have been met with at operation. For the purposes of our theme one of them needs be fully described.

CASE 3.—*A woman of middle age with blurring of vision of six months' duration supposedly accompanying the menopause. Primary optic atrophy, bitemporal hemianopsia with an unexpanded sella. Operative removal of small supra-*

5. Cushing and Davidoff: The Pathological Findings in Four Autopsied Cases of Acromegaly With a Discussion of Their Significance, Monogr. Rockefeller Inst. M. Research, No. 22, April 23, 1927, p. 43 (case III).

sellar chromophobe adenoma with prompt restoration of vision. Recurrence of symptoms after five months; operation repeated with equally successful result and return of menstruation. Second recurrence of symptoms after another eight months with third more radical operation, again with immediate improvement. Relief of symptoms to date, eighteen months.

Dec. 7, 1926: Admission of Mrs. Hansi G., an Austrian, 43 years of age, referred by Dr. H. O. Mosenthal of New York.

History.—The patient, a physician's wife and a highly cultured woman, had always enjoyed perfect health. The mother of five children, she had led a life of unusual activity and industry. Seven months before entry (*May, 1926*) her previously regular menses failed to appear: this was at first ascribed to preg-

Fig. 18 (case 3).—To show (natural size) the hazy but not definitely enlarged sella.

nancy and then to the menopause. Two months later (*July*) she became aware of haziness of vision in the temporal field of the left eye, associated with a slight feeling of supra-orbital discomfort; the left antrum was punctured, and the discomforts completely subsided. Vision in the left eye became progressively more clouded (central scotoma), and by November the temporal field of the right eye began to be affected.

Before her admission she had been thoroughly examined by a number of most competent specialists. There were no signals of disease other than the amenorrhea and disorder of vision; her weight (about 60 Kg.) had remained unchanged for years. The only definite opinions that had been expressed were: (1) by an ophthalmologist, that the trouble in view of the central scotoma on

the left, was unmistakably a retrobulbar neuritis most probably secondary to sinus disease or dental infection; (2) by a roentgenologist, that the sharpening of the anterior clinoid processes and evidences of some pressure absorption of the sella suggested a possible pituitary tumor.

Physical Examination.—This was wholly negative apart from (1) a possible slight pallor of the optic nerves more apparent in the left eye and (2) bitemporal field defects (fig. 17) with a relative central scotoma on the left where vision was reduced to 20/70 and a cleancut temporal hemianopsia on the right with 20/30 vision retained.

The sella (fig. 18) was of normal size and configuration, but in view of the existing symptoms of trouble in its neighborhood attention was called to the faint and somewhat irregular outlines which suggested mural absorption of the

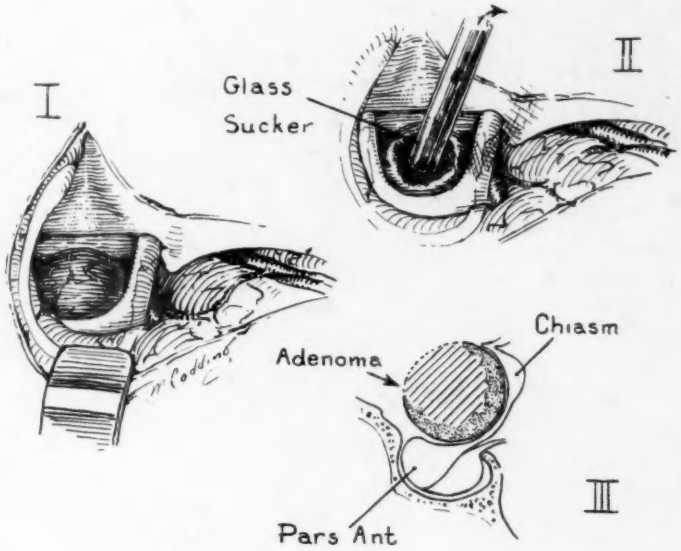


Fig. 19 (case 3).—Operative sketches showing: *I*, the position of the suprasellar adenoma which was covered only with arachnoid; *II*, the treatment of the lesion by suction, and *III*, the estimated size and position of the suprasellar tumor and the extent of the extirpation (shaded).

fossa from local compression. The olfactory sense was unaffected, and apart from the questionable menopause there were no secondary hypophysial or hypothalamic symptoms; the basal metabolic rate was within normal limits (—5 per cent before and + 5 per cent after the operation).

In the differential diagnosis, a meningioma was favored.

Operation I.—On *Dec. 13, 1926,* under procaine-ether anesthesia, the usual right frontal osteoplastic exploration was made. A yellowish tumor nodule covered only by arachnoid was found wedged in the embrace of the chiasm. On fully exposing these structures by stripping off the arachnoid, the nerves appeared to be elongated but not appreciably flattened. Apparently the pressure effect was being exerted directly against the anterior chiasm (fig. 19).

A few fragments of the tumor were taken for immediate fresh examination, and it proved to be an adenoma. The small nodule was then cleaned out so far as was thought safe by suction, leaving a cavity about a centimeter in diameter which, to be sure, still had adenoma in its walls.

Histologically the growth proved to be a typical chromophobe adenoma.

Postoperative Notes.—There was immediate subjective improvement in vision. On the day after the operation, to rough tests, it was evident that temporal vision had been largely restored in each eye. On the fifth day *(December 18)* the fields were found to be markedly widened, with 20/20 acuity on the right and improvement to 20/40 on the left. After a perfectly uneventful convalescence, the patient was discharged on *December 26,* with 20/20 vision in each eye and fairly well expanded temporal field peripheries (fig. 20). For nearly a week

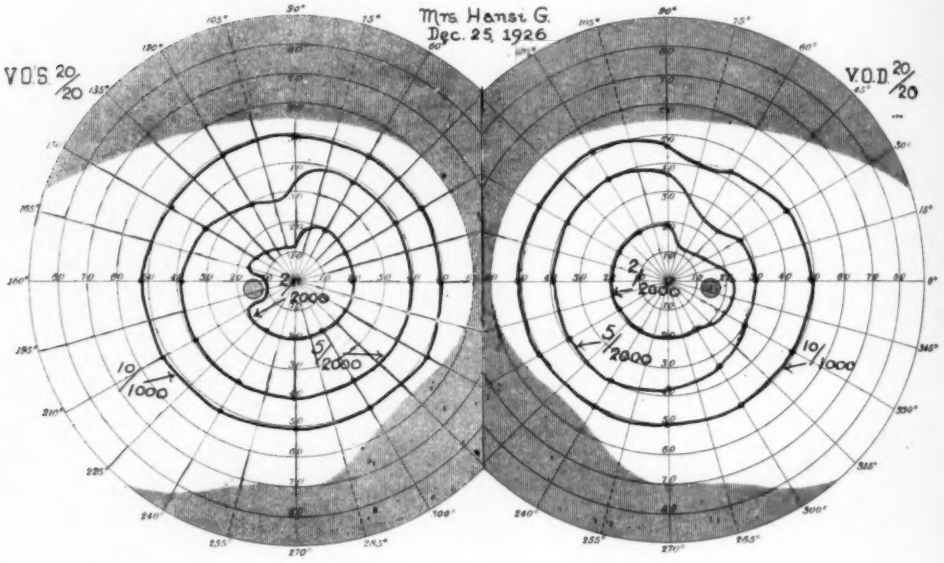


Fig. 20 (case 3).—Fields twelve days after operation for comparison with figure 17 (screen method only).

she had been able to read newspaper print and to do the finest needlework without any sense of ocular fatigue.

Subsequent Notes.—All went jubilantly for the next four months, when she again became conscious of periodic bitemporal haziness of vision.

May 2, 1927: The patient was readmitted to the hospital. The examination at this time showed essentially the same field defects (fig. 21) as on the first admission, though vision, to a 2 mm. disk on the screen, was wholly lost in the left eye, in which the acuity was reduced to 20/200. The pallor of the nerve head was possibly more apparent than on the previous admission. Evidently the adenoma had rapidly recurred, and with misgivings it was felt that the operation must be repeated.

Operation II.—On *May 7, 1927,* again under procaine-ether anesthesia, the region of the chiasm was reexposed far more easily than was expected. The

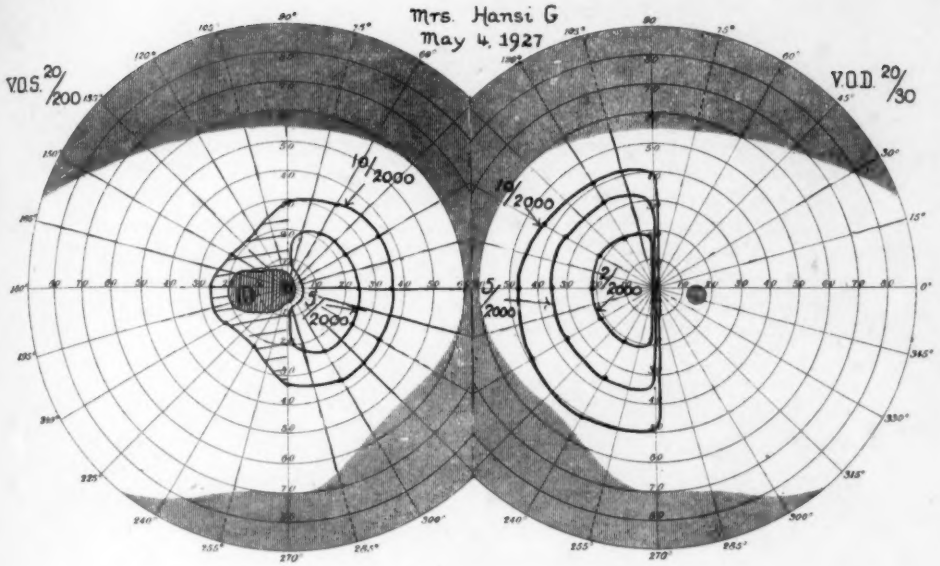


Fig. 21 (case 3).—Fields (screen method only) on patient's admission for recurrence of symptoms five months after first operation (cf. fig. 17).

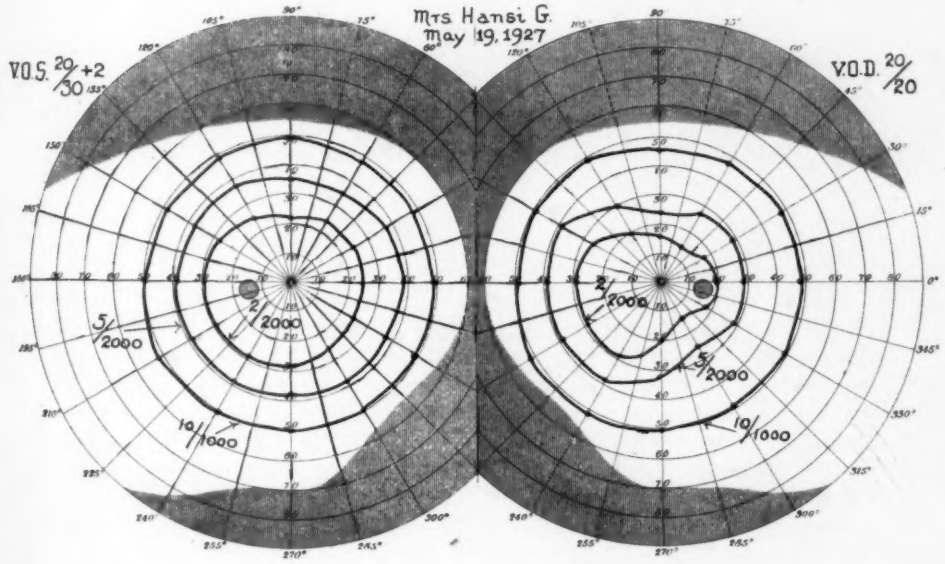


Fig. 22 (case 3).—Showing prompt postoperative widening of fields (cf. fig. 21) with restoration of essentially normal acuity for the second time (screen method only).

appearances of the soft, recurrent adenoma were almost precisely those seen at the first operation. It was attacked in the same intracapsular fashion as before (cf. fig. 19), and so far as could be told, the entire nodule, except a small fringe along the right optic nerve, was cleanly removed by suction.

Postoperative Notes.—As after the first operation, the patient was aware, on recovery from the anesthetic, of a vast improvement in vision. The wound healing was again perfect, and convalescence was uneventful except for a transient, mild polyuria which suggested some slight contusion of the infundibular stalk. At the end of a week *(May 14)*, the fields showed marked improvement though with some persisting temporal constriction in both eyes: vision in the left eye, 20/30; in the right eye, 20/20. At the time of her discharge twelve days after operation *(May 19)*, essentially normal peripheries had been regained in the temporal fields (fig. 22).

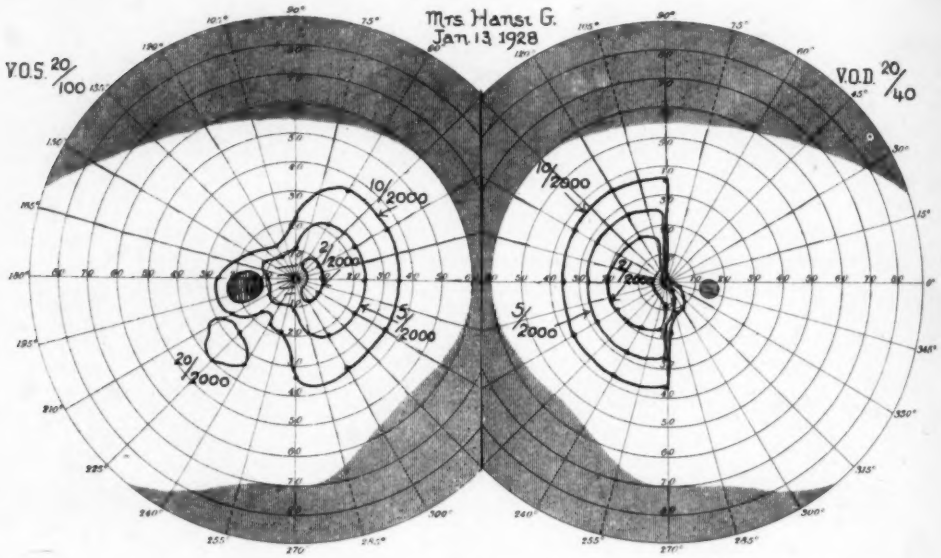


Fig. 23 (case 3).—Fields (screen method only) of Jan. 13, 1928, on patient's third admission owing to recurrence of chiasmal syndrome (cf. figs. 17 and 21).

Subsequent Notes.—Four months later *(Sept. 23, 1927)*, she reported that vision continued to be perfect and that for three months normal menstrual periods had been resumed after sixteen months of amenorrhea. In November the patient was under a great strain owing to her husband's illness, and she became once more aware, when sewing or reading, of an ominous haziness of vision in the left eye. Gradually this haziness grew worse, and two months later she again, for the third time, entered the hospital.

Third admission (Jan. 11, 1928).—The findings at this time were essentially those of the two preceding admissions. There were no evidences of pituitary dysfunction; her weight of 58 Kg. remained unchanged; the basal metabolic rate was within normal limits; normal menstrual periods had become reestablished. The pallor of the optic nerves, though not marked, was distinctly more apparent than on the previous admissions. The fields (fig. 23) showed essentially the same defects as before.

A third operation, rather than recourse to radiation, was suggested with hesitation, but the proposal was eagerly accepted by the patient.

Operation III.—On *Jan. 17, 1928,* the region of the chiasm was exposed for a third time (wholly under procaine anesthesia) with unexpected ease. Such filmy adhesions as were encountered were easily and bloodlessly separated by electrosurgical methods. At both of the previous operations the procedure had been limited to an intracapsular excavation of the adenoma, but it was possible at this session, after the small tumor had been partially excavated, to grasp its now somewhat thickened capsule and to dislodge it from its bed in the manner shown in the operative sketches (fig. 24). Only at one place, alongside the right optic nerve, did a fringe of the capsule remain adherent, and this fringe was

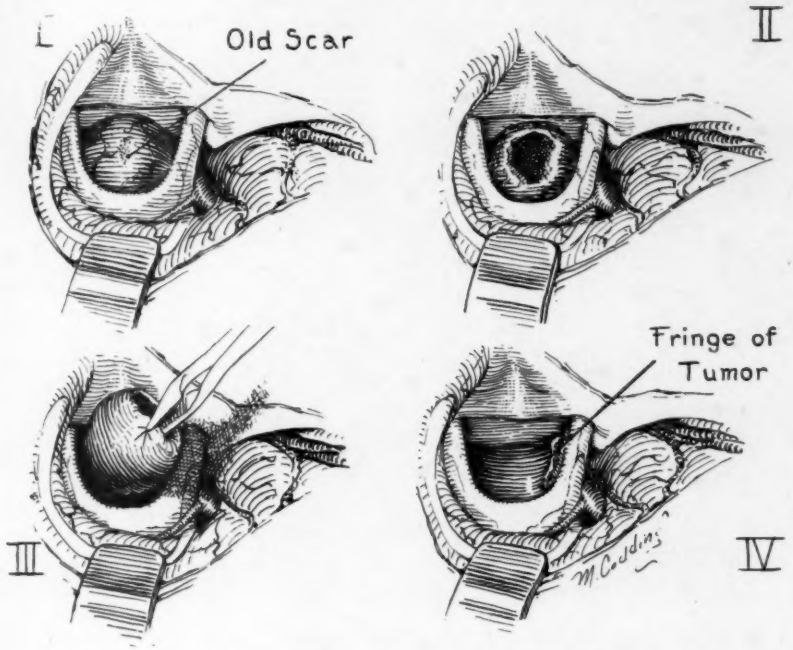


Fig. 24 (case 3).—Operative sketches of third operation, at which time: *I*, the scar of the preceding excavation could be seen; *II* and *III*, after partial excavation the nodule owing to its somewhat thickened capsule could be drawn out, leaving *IV* a small fringe of adenomatous tissue along the right optic nerve, which was coagulated.

electrically coagulated. In the base of the pocket could be seen what was taken for the pink upper surface of the hypophysis with no intervening dural diaphragm.

Postoperative Notes.—To the gratification of all, there was for the third time a prompt improvement in vision. The patient was sitting up on the second day, and by the fourth day *(January 21)* the acuity in the left eye had increased from 20/100 to 20/40; in the right, from 20/40 to 20/30 + 1. The left temporal field at this time had filled out more markedly than the right

(fig. 25), where there was a persisting upper defect which made us fear that the crossed fibers had been injured in the process of coagulating the fringe of tumor alongside the nerve.

Healing was again perfect (fig. 26), and at the time of discharge *(January 31)* two weeks after the operation, the acuity of the left eye was 20/30 and of the right 20/20. The nerve heads showed distinctly less pallor than before the operation; indeed, they might well have passed for normal. The clinical note states: "The patient has again been able to do the finest needlework without difficulty and has been amusing herself stringing minute Viennese beads to decorate the edges of a leather purse."

Subsequent Notes.—The patient was reexamined on *Aug. 1, 1929,* at which time the fields showed wider peripheries than those of *Jan. 31, 1928,* with vision

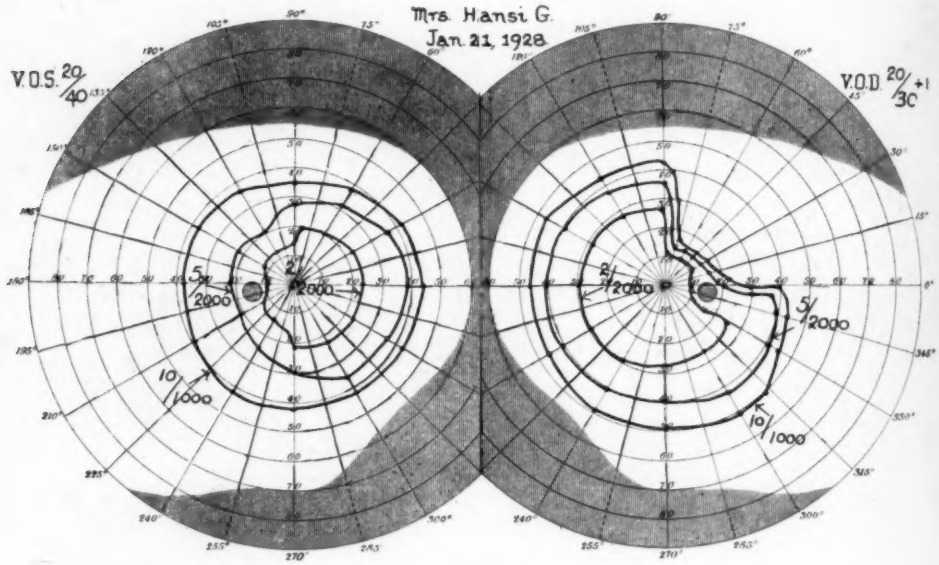


Fig. 25 (case 3).—Fields (screen method) five days after the third operation (cf. fig. 23).

in the left eye 20/30 and in the right eye 20/25. At the present writing, *(March 1, 1930),* over two years since the last operation, there has been no evidence of recurrence.

Comment.—From this long clinical history, several points may deserve special emphasis. The case shows: (1) that a syndrome well nigh indistinguishable from that of a suprasellar meningioma may be produced by a small suprasellar adenoma; (2) that relief to the encircling nerves and chiasm from pressure of the tumor may be followed by prompt restoration of essentially normal vision; (3) that if the small adenoma is not removed in its totality its continued growth may lead to a much earlier return of chiasmal symptoms than is usually seen after the partial surgical extirpation of a large intrasellar adenoma.

In comparing this somewhat unusual case with other similar ones in our series of suprasellar adenomas, it is evident that in the differential diagnosis insufficient emphasis has been laid on the fact that the sella, though of essentially normal contour, has shown a greater degree of pressure absorption than has accompanied any of the suprasellar meningiomas. Two other cases with a similarly erroneous preoperative diagnosis may be briefly recorded.

CASE 4.—*A mining engineer, 50 years of age and of vigorous health, entered the clinic on July 2, 1928, with the following history. In 1924, he was found to have a temporal hemianopsia in the left eye. In spite of the usual preliminary*

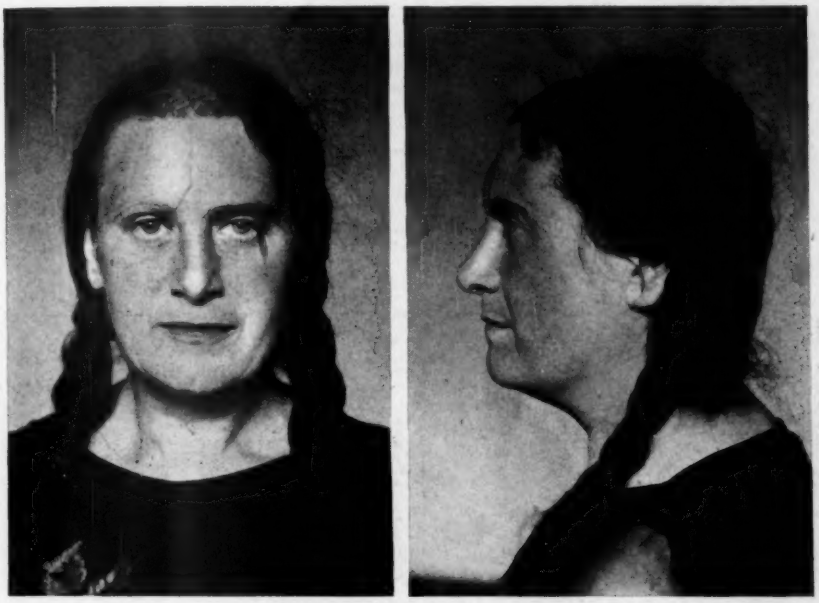


Fig. 26 (case 3).—Inconspicuous scar after repeated transfrontal operations for recurrent suprasellar adenoma.

intranasal operations, tonsillectomy and the extraction of supposedly infected teeth, the loss of vision progressed, and in the course of the next two years the left eye had become practically blind. Meanwhile a similar process had begun slowly to affect the right eye.

Examination.—Examination showed: (1) marked atrophic pallor in the left eye where bare perception to light was retained; (2) typical temporal hemianopsia (fig. 27) in the right eye with acuity of 20/70; (3) an unexpanded sella showing evidence however of pressure atrophy (fig. 28.) The basal metabolic rate was —22 per cent.

A presumptive diagnosis was made of suprasellar meningioma, though adenoma was considered a possibility.

Operation.—At operation, on *July 11, 1928,* an encapsulated suprasellar adenoma of chromophobe type was encountered and radically removed, chiefly

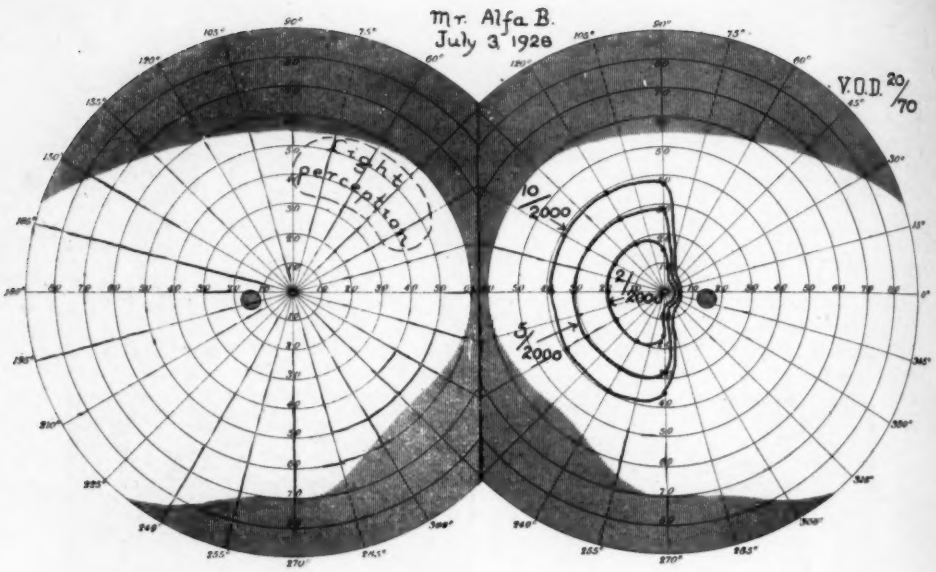


Fig. 27 (case 4).—Fields (screen method only) on patient's admission after four years of a progressive chiasmal syndrome due to a suprasellar adenoma.

Fig. 28 (case 4).—Showing atrophic sella of essentially normal configuration in association with a suprasellar adenoma erroneously diagnosed as probable meningioma.

by suction. An uneventful surgical recovery followed, but there was no immediate postoperative change for the better in the eyes.

The patient has resumed his business in Manila, and, according to his last report of *June, 1929,* there has been a slow progressive improvement in vision, and he has regained the ability to recognize objects even with his previously blind left eye.

The brief abstract of another case, similar to the foregoing one, though with a more favorable surgical outcome, follows. Here again the chiasmal syndrome was looked on as favoring a meningioma owing to the unexpanded sella, insufficient significance having been ascribed to its atrophic condition.

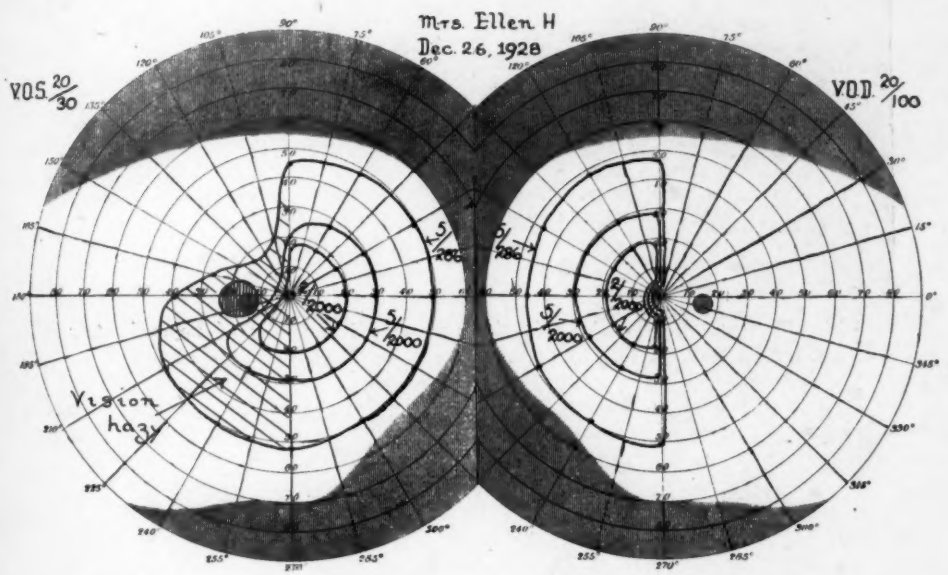


Fig. 29 (case 5).—Fields (combined screen and perimeter) on patient's admission.

CASE 5.—*A married woman 40 years of age was referred to the clinic because of failing vision and with the diagnosis of suprasellar tumor. For seven months there had been a gradual bitemporal loss of vision more advanced in the right eye.*

Examination.—Examination showed: (1) definite slight pallor of the disks; (2) bitemporal field defects (fig. 29), with vision in the left eye 20/30 and in the right eye 20/100; (3) a pituitary fossa that was unenlarged though giving evidence of pressure absorption (fig. 30). The history and the physical examination were otherwise negative. The basal metabolic rate was + 3 per cent. A suprasellar meningioma was suspected.

Operation.—The operation *(Jan. 7, 1929)* was conducted wholly under local anesthesia. The anterior chiasmal crotch was found to be expanded by a suprasellar chromophobe adenoma which was radically attacked by electrosurgical methods and suction until its capsule was wholly collapsed, leaving the optic nerves quite freed from contact. During this procedure, owing to a sudden movement on the part of the patient, the right optic nerve was inadvertently

Fig. 30 (case 5).—To show (slightly retouched) the sella of approximately normal outline but atrophied by pressure.

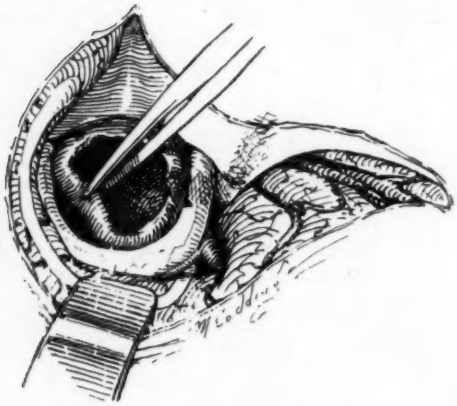


Fig. 31 (case 5).—Operative sketch showing accidental dehydration cut in right optic nerve; also process of withdrawing and shrinking of emptied tumor capsule by coagulation current.

touched by the needle carrying the dehydrating current. This left a notch (fig. 31) in the inner side of the nerve, and it was feared that the hemianopsia of this eye would thereby have been rendered permanent.

There were no postoperative complications, and the usual perfect wound healing was attained. It was evident on the third day that there was some return of vision in the previously blind temporal field of the right eye and that to large objects normal peripheries had been regained in the left. The extent of widening of the fields at the time of the patient's discharge is shown in figure 32.

A year later (*Feb. 22, 1930*) the condition remained stationary, satisfactory and useful vision having been well retained.

These examples will suffice to show how closely the syndrome of a suprasellar adenoma may simulate that of a meningioma. Still

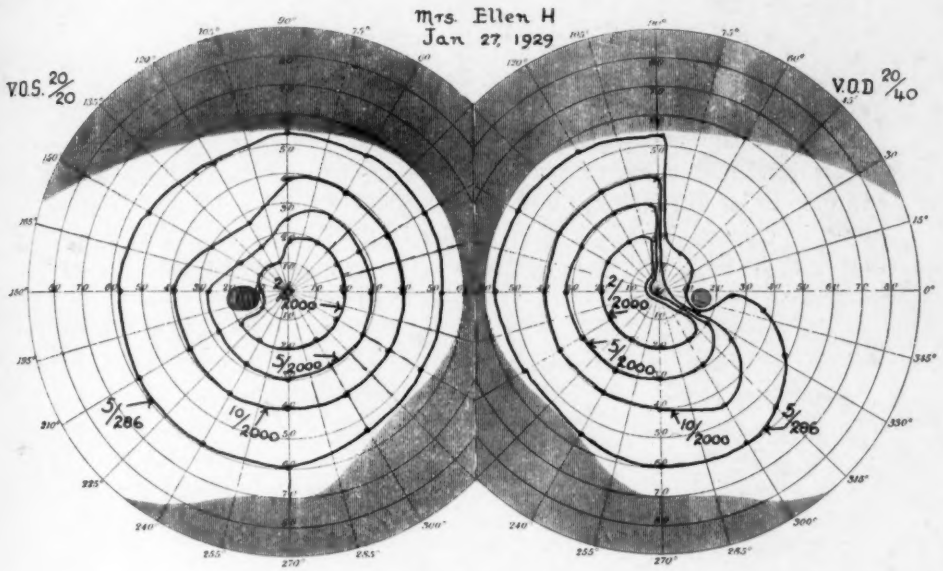


Fig. 32 (case 5).—Fields (combined screen and perimeter) three weeks after operation, for comparison with figure 29.

better illustrations will, in all probability, have come to the attention of other neurosurgeons. My one-time assistant, Mr. Norman Dott of Edinburgh, writes that in operating on a man 67 years of age with a typical progressive chiasmal syndrome and a normal sella, he found a cyst that presented in the anterior crotch of the chiasm. This cyst was mistaken for a Rathke's pouch lesion until sections of its wall came to be microscopically examined, when it proved to be a small cystic adenoma.

On two or three occasions I have been led to make a definite preoperative diagnosis of a Rathke's pouch tumor on the basis of suprasellar shadows due to calcification and have found a partly

calcified adenoma, but this is exceedingly rare [6]—even more rare than for a psammomatous suprasellar meningioma to cast discernible shadows, though an example of this was given in case 10 of our former paper.

In the differential diagnosis between adenoma and meningioma, the points to be emphasized are: (1) the greater evidence of pressure atrophy of the sella in the former; (2) the coexistence of mild hypopituitary signs, and (3) the more rapid progress of the symptoms, for a pituitary adenoma is doubtless a more rapidly expanding tumor than a meningioma. Still, there must be exceptions to this rule, and in one of the cases cited (case 4) the symptoms were of four years' duration, though, to be sure, they were far advanced (cf. fig. 27).

CRANIOPHARYNGIOMAS

This term may be advantageously employed to connote the protean group of congenital tumors that arise from developmental faults in closure of the hypophysial duct. They are variously called craniopharyngeal duct cysts, tumors of Rathke's pouch, adamantinomas, and so on. Being congenital, they usually give first evidence of their presence in childhood, and since lime salts in the great majority of cases come to be deposited in these tumors, the roentgenologist may be the first to make a definite pathologic diagnosis.

The presence of suprasellar calcification speaks strongly in favor of one of these lesions, the amount of calcification varying from an enormous mass the size of a tennis ball to the presence of a few flecks, the exact suprasellar position of which can be detected only by stereoscopic roentgenograms. One cannot, however, in arriving at a clinical diagnosis, rely unequivocally on these suprasellar shadows, for, as just related, they may be cast by psammomatous depositions in a suprasellar meningioma; calcification, indeed, may, in rare instances, be present in an adenoma.

These craniopharyngiomas are far more common than is generally supposed. Our series contains, to March 1, 1930, eighty-seven histologically verified examples with many more unverified ones that have not been regarded as amenable to surgical intervention. The tumors may vary from a thin-walled, relatively silent cyst the size of a pea to an enormous solid and calcified or multicystic tumor as large as a fist, with secondary hydrocephalus and all manner of intracranial symptoms. In addition to their frequent chiasmal symptomatology, they may cause secondary pituitary and hypothalamic disturbances of the most varied sort, depending on the primary seat of the lesion, the size which it attains, the part of the brain chiefly affected and the age of the patient at the time the tumor begins to cause pressure symptoms.

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6. Deery, E. M.: Calcification in Pituitary Adenomas, Endocrinology **13**:455-458, 1929.

If the "anlage" of the growth happens to lie within the sella, the latter expands, and the symptoms may resemble those of an intrasellar adenoma. If it lies above the sella, the chiasmal syndrome may be paramount, and the sellar outlines relatively normal. The symptoms may remain relatively stationary for many years and may not appear until adult life; on the other hand, for reasons unapparent, the growth may become active and attain a huge size in the early years of life.

When symptoms of the lesion appear before adolescence and when, as commonly occurs, the anterior pituitary body fails properly to develop, variable degrees of sexual and skeletal infantilism are produced. When, on the other hand, symptoms are delayed till adult life, constitutional signs of secondary hypopituitarism may appear with sexual dystrophy, a low basal metabolic rate, and so on. And should the growth happen to be so situated as to involve the nerve centers in the tuber cinereum at any age, varying grades of polyuria, hypersomnia and adiposity may occur.

In spite of these most variable and protean constitutional effects produced by craniopharyngiomas, this tumor of all intracranial tumors is ordinarily the easiest to diagnose. However, when the sella is undeformed, when there are no suprasellar shadows and, in the absence of pituitary manifestations, when the symptoms of a craniopharyngioma are postponed to adult life, one of these tumors may be well nigh impossible to distinguish clinically from a suprasellar meningioma.

For the purposes of this paper two illustrative cases have been selected.

CASE 6.—*Progressive failure of vision for twelve months in a woman approaching middle age. Bitemporal hemianopsia with questionable optic atrophy. Intercurrent nasal operations. Essentially normal though atrophic sella. Clinical diagnosis—probable suprasellar meningioma. Evacuation and partial removal of small suprasellar craniopharyngeal pouch cyst. Postoperative polyuria. Improvement in vision. Unaccountable death two months later.*

Nov. 3, 1928: Mrs. C., a Jewess, 40 years of age, on the recommendation of Dr. John M. Wheeler of New York, was admitted to the hospital with the presumptive diagnosis of suprasellar meningioma.

History.—The patient, an intelligent and cooperative woman, had been subject to frequent "nervous breakdowns," and during the course of her life had had several surgical illnesses—an empyema requiring drainage in childhood, a ventral suspension with appendectomy in 1913, and a mammary abscess in 1915 during one of her several pregnancies.

In *November, 1927,* in an automobile accident she received a blow on the forehead followed by epistaxis and orbital ecchymosis. To this accident she ascribed her present trouble, which began soon after with blurring of vision in the left eye.

On *Jan. 6, 1928,* she first consulted Dr. Wheeler, who found faulty color perception in the left eye and a lower temporal field defect. Several radical intranasal operations were subsequently performed (septal resection, opening of

both antra, the sphenoid and ethmoid cells), leaving her with a perforated septum and a chronic nasal discharge. She was subsequently given a course of antisyphilitic treatment.

By *October, 1928,* when she was again examined by Dr. Wheeler, a left hemianopsia was present, and the temporal field of the right eye had begun to be subjectively impaired. After consultation with Dr. Foster Kennedy, she was referred to the Brigham Hospital.

Physical Examination.—The patient was a healthy appearing Jewess of middle age complaining of loss of vision, constant nasal discharge and persistent frontal headache. Apart from the ophthalmologic findings and evidences of preceding nasal operations, the examination was wholly negative.

She was found to have a complete bitemporal hemianopsia, to small disks as well as to colors, by the screen method, and by the perimeter a lower bitem-

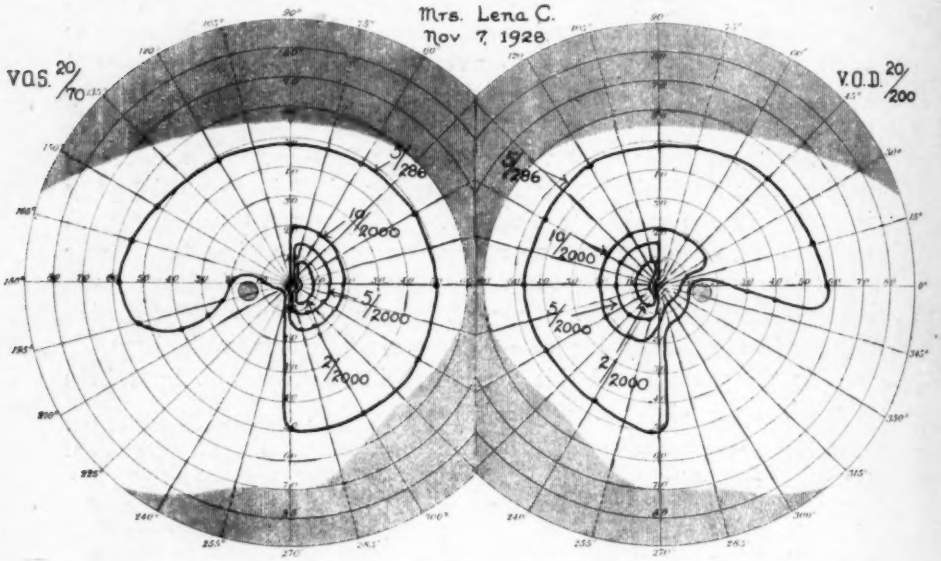

Fig. 33 (case 6).—The fields (perimeter and screen) on admission of a patient with craniopharyngioma.

poral quadrantal defect was disclosed (fig. 33). Vision in the left eye was 20/70; in the right eye, 20/200. In spite of her greatly impaired vision, the nerves ophthalmoscopically showed only a questionable evidence of pallor. The sella was of normal size, but thin-walled and with sharpened clinoid processes (fig. 34). A single minute fleck of calcification lying between the anterior clinoid processes was detected and recorded by the roentgenologist, Dr. Sosman, but it did not suffice to justify any attempt to differentiate between craniopharyngeal pouch cyst and meningioma.

The basal metabolic rate was —6 per cent. Menstruation was regular and normal. There had been no change in weight, which for years had averaged 60 Kg. The olfactory sense was unimpaired. There was a mild polyuria, and she had an extremely low blood pressure averaging 100 systolic over 70 diastolic.

In agreement with Drs. Kennedy and Wheeler, the diagnosis of a suprasellar meningioma was favored, although the thinning of the sella and the field defects were out of all proportion to the opthalmoscopic appearance of the disk.

Operation.—Nov. 19, 1928. The procedure was conducted wholly under local anesthesia. As the dura was not adherent, the bone flap was more easily reflected than in most cases of meningioma. A small chiasm of normal appearance was readily exposed. There was no evidence of tumor until, after the arachnoid had been thoroughly stripped away, a small cystic protrusion was finally detected in the depths between the legs of the chiasm (fig. 35). A suction needle was inserted into this protrusion, and about 6 cc. of highly turbid fluid rich in cholesterin crystals was withdrawn. Though there was small space in which to work, the collapsed cyst wall was picked up and partly dislodged; but since its deeper portion proved to be adherent, presumably to the tuber, it was considered unwise to attempt its total removal, and only a small portion was taken for histologic identification

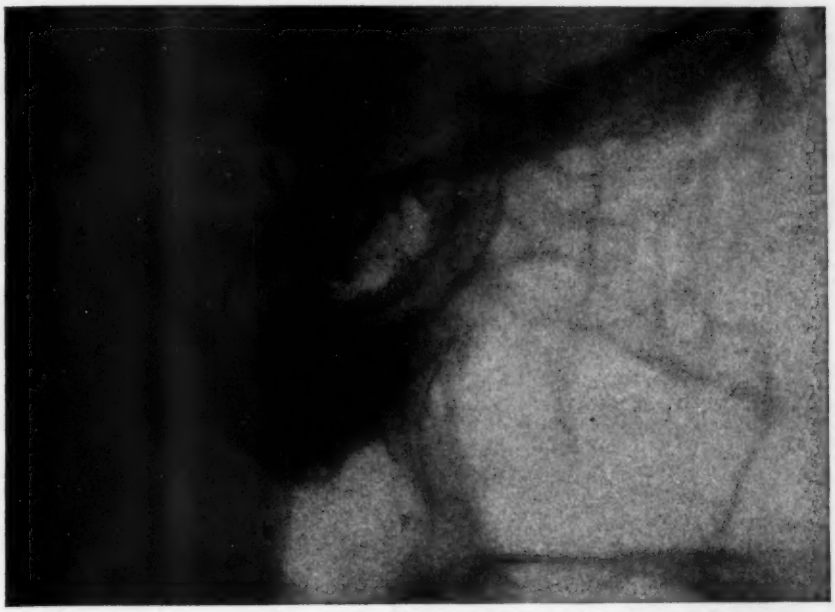

Fig. 34 (case 6).—Showing the unexpanded sella with sharpened clinoid processes.

(fig. 36). After careful hemostasis, the flap was replaced and the wound sutured in the usual painstaking detail.

Postoperative Course.—The course was unexpectedly stormy. There was considerable headache for a few days, and the formation of a postoperative clot was feared. Owing doubtless to the manipulation of the cyst, the preexisting mild polyuria was greatly exaggerated, amounting to 12 liters in the first twenty-four hours, with gradual subsidence to about 5 liters up to the time of the patient's discharge. Injections of "pituitrin" controlled the polyuria, but the patient strongly objected to them.

In other respects she did reasonably well. Although the crossed fibers to the right eye had evidently been damaged by the operative manipulations so that a right temporal hemianopsia was complete, the vision in this eye improved from 20/70 to 20/40. In the left eye the fields opened out widely even to small disks

Fig. 35 (case 6).—Operative sketch, showing small deep-lying cyst in crotch of normal-appearing chiasm.

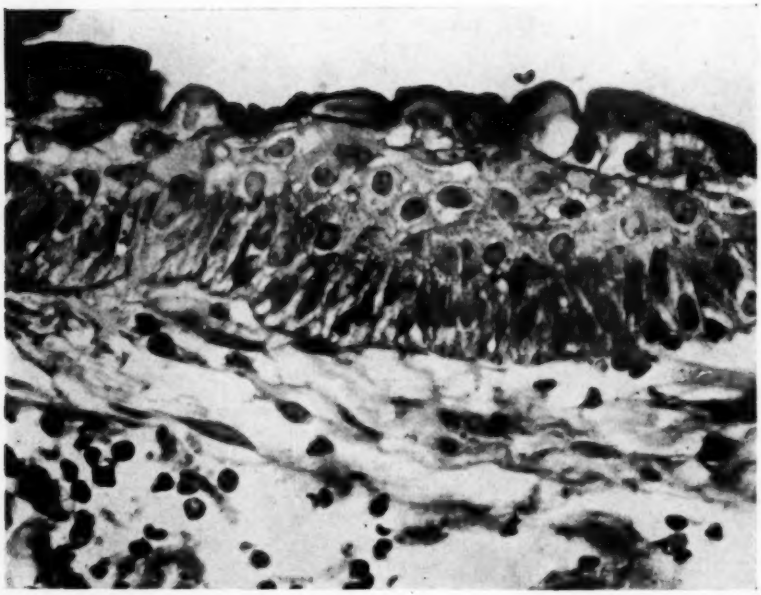

Fig. 36 (case 6).—Photomicrograph of cyst wall of pharyngeal duct anlage (phosphotungstic acid-hematoxylin; × 600).

(fig. 37), and practically normal vision was regained. Wound healing had been perfect and on *Dec. 18, 1928,* after a prolonged hospital sojourn, she was finally discharged in apparently good condition in spite of her mild residual polyuria.

Two weeks later she was admitted to a hospital in New York with vomiting, fever and a low blood pressure. She died on *Jan. 14, 1929,* from an undetermined cause. There was no postmortem examination.

Comment.—The importance of the record from an ophthalmologic standpoint lies in the fact that a subjective disturbance of vision had been present for a year and temporal field defects for ten months before the operation, and yet the eyegrounds would have passed as essentially normal. Professor Derby was asked to examine the fundi, and he

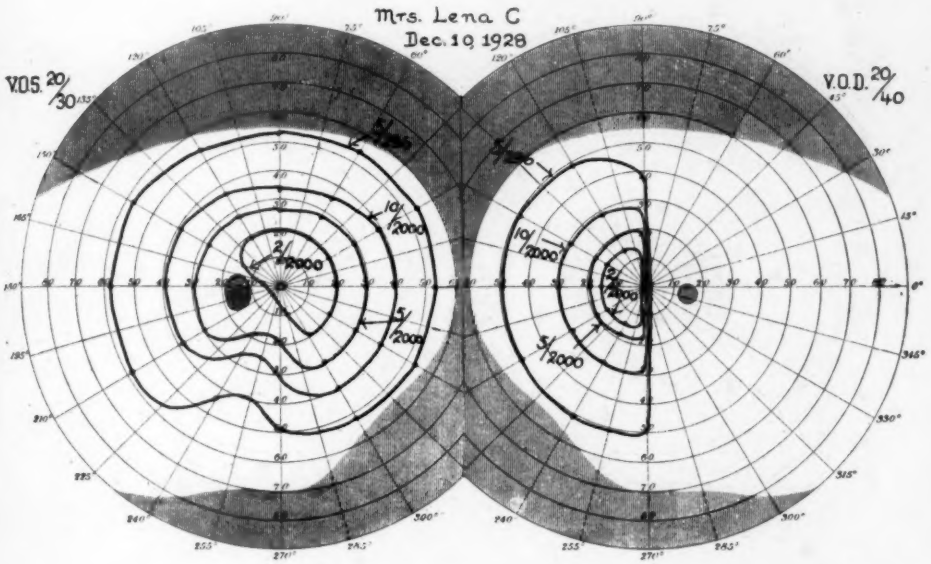

Fig. 37 (case 6).—Patient's fields (perimeter and screen) after evacuation of small cystic craniopharyngioma (cf. fig. 33).

stated that in view of the history he could "imagine a beginning atrophy" in the nerves of either side which possibly was more marked in the left, but without having seen the disk for purposes of comparison before the change occurred, he would be inclined to regard them as normal.

In retrospect, a correct preoperative diagnosis of a craniopharyngioma should possibly have been made in this case on the basis of the minute fleck of calcification that was detected on the stereoscopic films. However, the patient was in the meningioma rather than in the craniopharyngioma age, and suprasellar meningiomas, as already stated, may occasionally be sufficiently psammomatous to give shadows that are even more marked than those detected by Dr. Sosman in this patient's films.

From the want of a postmortem examination, what may have been the cause of death, whether the spread of infection from the chronically discharging ethmoids or the rapid refilling of the cyst with a fatal hypothalamic disturbance, is impossible to say. Nevertheless, the history of this unfortunate woman serves to illustrate the hazards of operations for craniopharyngeal duct tumors if more is attempted than the mere evacuation of a cyst. Experience has shown that these cysts invariably refill after operation, and though this, in favorable cases, may be a matter of years, it is more often one of months, so that the temptation to remove as much of the cyst wall as possible is irresistible.

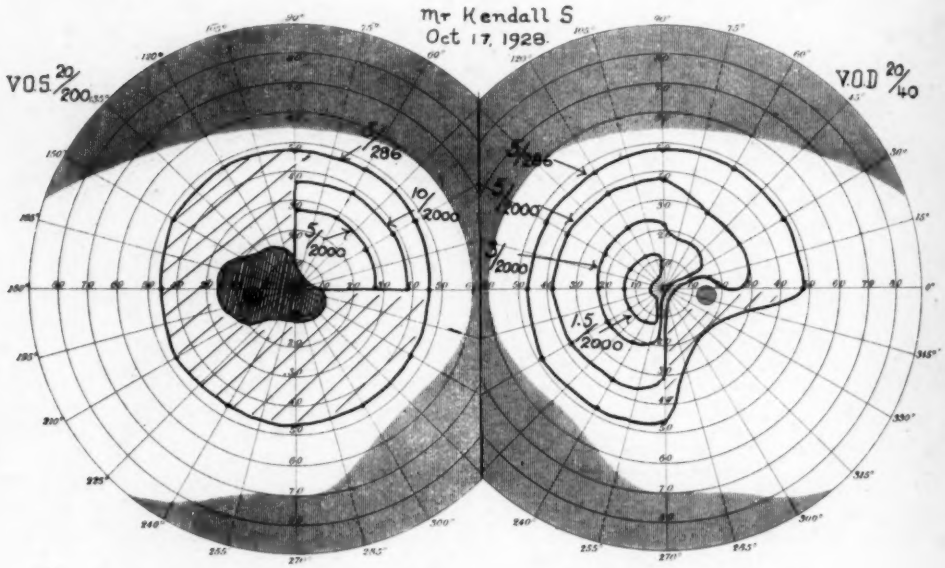

Fig. 38 (case 7).—Patient's fields (screen and perimeter) on admission.

If, on the other hand, the tumor should be largely solid and should at the same time underlie an anteriorly placed and short-legged chiasm, the only possible way to expose it is deliberately to commit the patient to a permanent bitemporal hemianopsia by an anteroposterior transsection of the chiasm, as was done in the following case.

CASE 7.—*Progressive failure of vision for six months in a man in early middle age. Bitemporal field defects with primary optic atrophy and a normal sella. Personality changes with hypersomnia and polyuria. Diagnosis: meningioma or glioma involving chiasm and third ventricle. Operation with partial removal of a suprasellar craniopharyngioma after chiasmal transection. Subsequent classic bitemporal hemianopsia with improvement in vision.*

Oct. 3, 1928: Mr. K. K. S., a university teacher, 46 years of age, referred by Dr. Lewis B. Paton of Providence, R. I., for failure of vision, was admitted to the hospital.

History.—The patient, a man of exceptionally good health and habits, for six months had been losing sight, in association with a tendency to undue drowsiness, fatigability, unnatural thirst, a loss of sexual activity, a gain of 20 pounds (9 Kg.) in weight and definite personality changes.

Examination.—The ophthalmologic findings showed: (1) a slight questionable pallor of the disks in an otherwise normal retina; (2) marked field defects evidently bitemporal in character (fig. 38) far more advanced in the left eye, where there was a large central scotoma. Vision in the left eye was 20/200 with difficulty; in the right eye 20/40. The sella was normal, with no trace of any regional calcification (fig. 39). The basal metabolic rate was —32 per cent. There

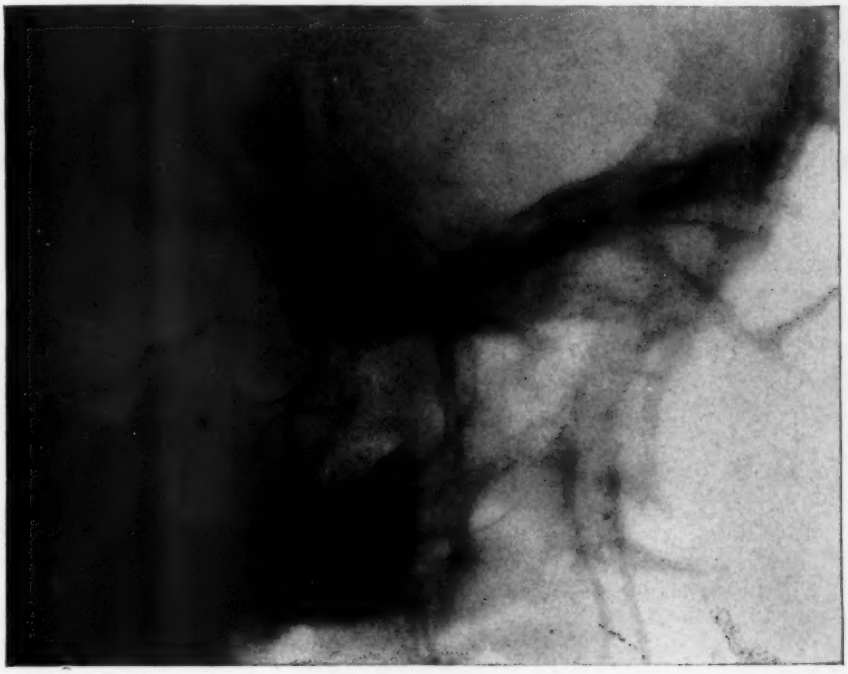

Fig. 39 (case 7).—Sella of normal configuration.

was a polyuria of about 4 liters daily, with associated moderate polydipsia. The temperature tended to be subnormal.

In addition to these objective symptoms, the patient showed considerable mental instability. He was continually drowsy, and when aroused somewhat euphoric, occasionally incontinent, subject to periodic olfactory hallucinations and wholly indifferent to his plight.

Diagnosis.—A tumor of the third ventricle affecting the chiasm was at first suspected. Accordingly, on *October 25* ventriculograms were made, but they showed no ventricular abnormality. However, on the chance of there being a small third ventricle tumor, he was given a course of deep radiotherapy without symptomatic improvement. To clarify the diagnosis and in the vain hope that there might possibly be a large suprasellar meningioma, an exploratory operation was advised.

Operation.—On *Nov. 5, 1928,* under procaine-ether anesthesia, the usual right frontal osteoplastic flap was reflected. A large amount of fluid was secured on opening the dura, and a normal-appearing, short-legged chiasm was finally brought into view. What was taken to be the anterior end of a Rathke's pouch tumor, because of a few yellowish flecks on its surface, could be seen (fig. 40, *I*) in the depths of the anterior chiasmal crotch. A bit of this tissue was taken for immediate histologic examination, and it proved to be composed of epithelial cells.

Somewhat at a loss to know what more to do, the arachnoid attachments were stripped away so as to give a full view of the chiasm, the posterior part of which was found to be elevated, deeply grooved by the right anterior cerebral artery, and with a bulging mass pushing forward through the thin wall of the third ventricle (fig. 40, *II*). A needle was inserted into this retrochiasmal protrusion, and about a cubic centimeter of discolored fluid, turbid with cholesterin crystals, was secured.

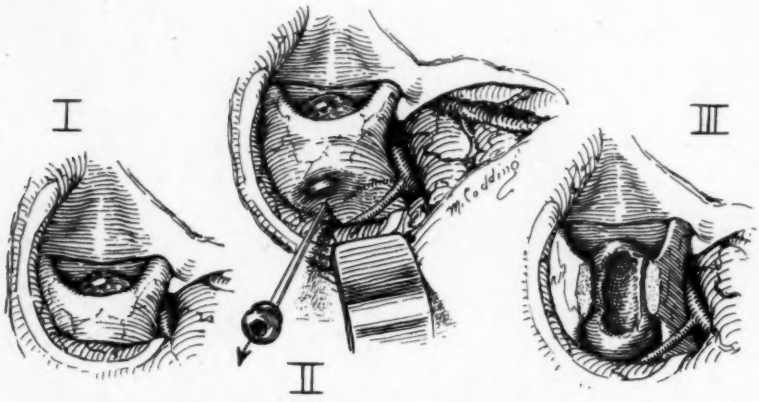

Fig. 40 (case 7).—Operative sketch to show: *I*, the primary view of a relatively prefixed chiasm almost wholly concealing a craniopharyngioma; *II*, puncture of retrochiasmal cyst; *III*, transection of chiasm with exposure of tumor.

There seemed to be only one chance of preserving at least binasal vision for any length of time, and that was by transecting the chiasm. It was hoped, moreover, that this procedure might possibly expose the lesion sufficiently well to permit of its partial extirpation. Accordingly, the chiasm was electrically divided, and, as shown in the sketch (fig. 40, *III*), the nerves promptly separated so that they were no longer under tension; the reddish tumor, which presented in the midline, was partly excavated.

The growth proved to be a typical craniopharyngioma (fig. 41).

Subsequent Course.—The operation was followed by a distinct improvement in vision, especially in the left (the poorer) eye, in spite of the surgically produced bitemporal meridianal hemianopsia, the scotoma so far as it affected the uncrossed macular bundle having largely disappeared (fig. 42). Wound healing was perfect, and the patient was discharged on *Nov. 26, 1928.*

Encouraged by the marked improvement in vision, the patient made an effort, after the first of the year, to resume his collegiate activities and former contacts, but this proved too much for him, particularly because of his tendency to fall

asleep and to periods of considerable mental confusion.[7] Fortunately, in these respects he showed a distinct turn for the better after a few months had passed. The temporarily improved vision in the left eye, however, had begun to fail during the winter, and on *July 1, 1929,* when he was last examined, the vision in the left eye had again receded to 20/200, and the seeing field was once more restricted to the upper nasal quadrant. The condition in the right eye, in which reading vision was retained, remained unaltered.

Here again was a congenital suprasellar lesion that had given no symptoms until adult life, when in the presence of a normal sella evidences of chiasmal involvement had appeared with considerable abruptness. The primary chiasmal syndrome, to be sure, had become overlain and obscured by a secondary hypothalamic syndrome, but the

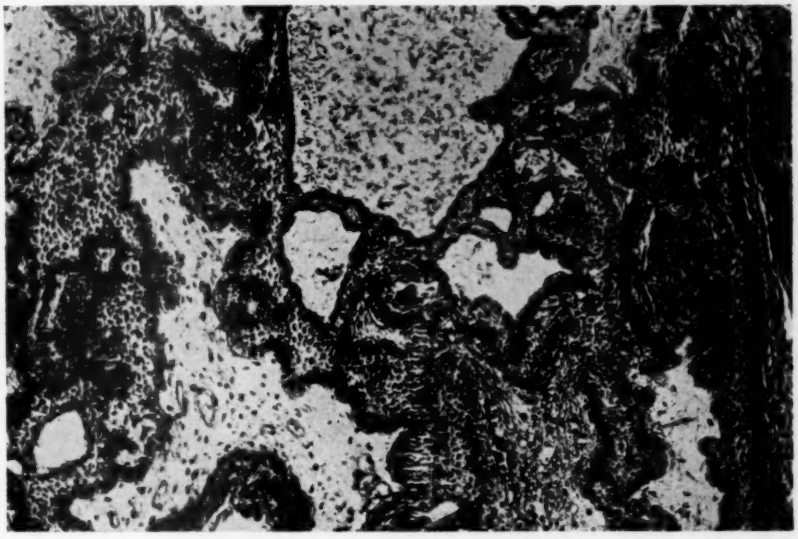

Fig. 41 (case 7).—Photomicrograph of noncalcified tumor of Rathke's pouch (phosphotungstic acid-hematoxylin; × 110).

case nevertheless has been chosen, out of many, for the purposes of this report since it illustrates the inherent difficulties of dealing with these particular tumors.

It would carry this paper far beyond its proper scope to dwell in any great detail on the surgical treatment of the craniopharyngiomas. One might suppose that congenital lesions of this kind could be readily

7. Apart from the hypersomnia, there were no other recognizable hypothalamic symptoms. The polyuria had disappeared, and he had no further tendency to adiposity, having lost the excess 20 pounds present on his admission to the hospital. The patient survived with vision retained in the right eye until his death on Nov. 26, 1929. A postmortem examination disclosed an uncalcified tumor measuring 3 by 2 by 2.5 cm. filling the third ventricle.

enucleated, but as a matter of fact, even when of small size and easily accessible to view, they are so adherent to chiasm and third ventricle that in dislodging them these delicate structures may be severely damaged. When, on the other hand, the chiasm is of the prefixed type so that the tumor is completely concealed, as in the case of this patient, the problem is vastly more difficult.

Dr. Oljenick of Amsterdam has for the past two years been making a study of eighty-four histologically verified craniopharyngiomas in the Brigham Hospital series. His report will deal with the subject from all standpoints—developmental, clinical, pathologic and surgical—but it may not be out of place to call attention at this time to a few points

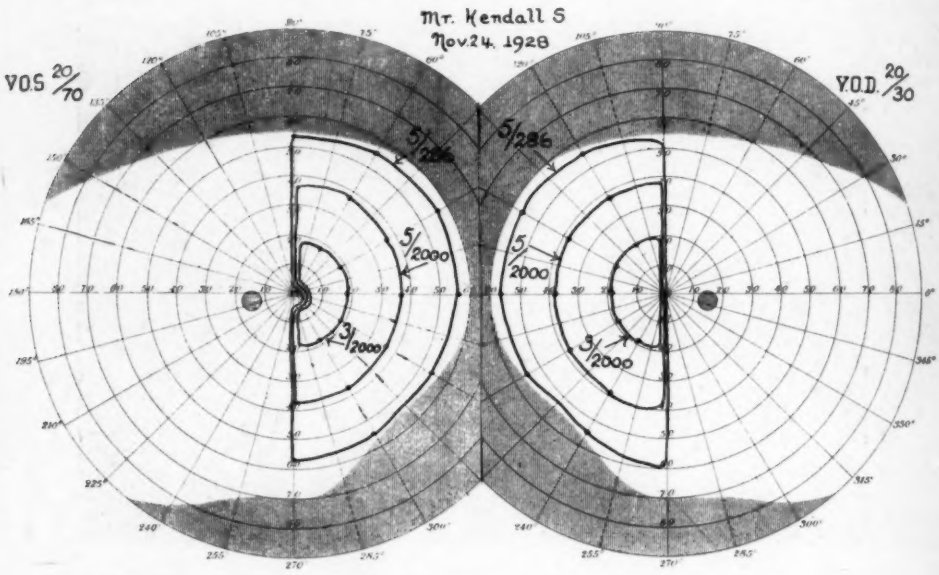

Fig. 42 (case 7).—Fields of vision three weeks after transection of chiasm to expose suprasellar craniopharyngioma, for comparison with figure 39.

chiefly of ophthalmologic interest. Brief reference to a single case may serve as a basis for what may be pertinent to our present topic.

CASE 8.—*A young woman 19 years of age who had never menstruated and was somewhat obese was admitted to the clinic because of headaches and failing vision. She was found to have (1) bilateral choked disk of 5 diopters superimposed on primarily atrophic (?) nerves; (2) a right homonymous hemianopsia with vision in the left eye 20/200 and in the right eye 20/70; (3) a malformed sella with shadows of a calcified mass above and behind, and an undeveloped dorsum sellae, the anterior clinoid processes being normal (fig. 43). The basal metabolic rate was —31 per cent.*

A diagnosis of Rathke's pouch tumor with impaired hypophysial function and hydrocephalus was unquestioned.

While under observation she had an attack of pressure syncope which was overcome by the administration of hypertonic salt solution while preparations were made for an emergency operation in the hope of finding a cyst that could be evacuated.

At the operation, after puncturing the dilated ventricle to permit elevation of the frontal lobe, the conditions shown in the illustration (fig. 44) were found. There was a bulging prefixed chiasm, widely expanded by an underlying and wholly concealed tumor, with the optic tracts deeply cut by the encircling anterior cerebral arteries. Only on this basis could the homonymous hemianopsia be explained, for the lesion appeared to be symmetrically disposed. The right

Fig. 43 (case 8).—To show a deformed and tilted though undistended sella with normal anterior clinoids and rudimentary dorsum surmounted by the shadows of a calcified craniopharyngioma which lay behind a prefixed chiasm (cf. fig. 46).

anterior cerebral artery was divided between clips, the ends of the divided vessel immediately separating fully a centimeter as the constriction was relieved.

Back of the chiasm was a bulging bluish cyst which was punctured, and 10 cc. of typical "motor-oil" fluid rich in cholesterin crystals was sucked out. A few bits of the cyst wall were removed for histologic verification of the lesion, but no effort was made further to expose the tumor or to attempt its removal, and the osteoplastic flap was replaced.

There was a temporary marked postoperative polyuria (10 liters), easily controlled by posterior pituitary extracts, which soon subsided to normal. There

was early subsidence of the choked disks, and the fields of vision promptly widened out to their normal peripheries with an acuity of 20/50 in each eye. The patient was discharged to her home in excellent condition, but two months later the symptoms all reappeared with evidence of acute hydrocephalus, which proved fatal.

This case, though not an example of the chiasmal syndrome under discussion, nor one which offered any difficulties of preoperative diagnosis, has been brought into this discussion as it will illustrate certain matters that cannot fail to be of concern to ophthalmologists. It shows in the first place that a choked disk may come to be superimposed on a primary atrophy when a suprasellar tumor finally causes a secondary hydrocephalus from occlusion of the foramina of Monro. Such a thing, however, could happen only with a posteriorly placed tumor such as was

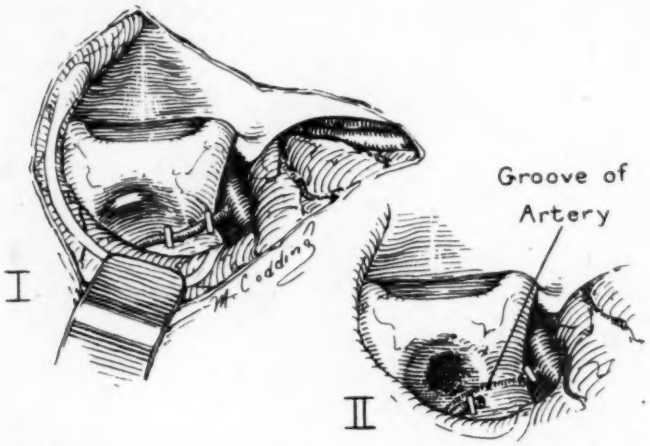

Fig. 44 (case 8).—Operative sketch, showing: *I*, a prefixed chiasm overlying a craniopharyngioma; note the right optic tract deeply cut by anterior cerebral artery which has been doubly clipped preparatory to section; *II*, withdrawal of ends of divided artery after section with exposure of groove in the nervous tissues.

present in this case, there being no direct pressure on the optic nerves to prevent access of cerebrospinal fluid to their sheaths. With a suprasellar meningioma, even one large enough finally to cause hydrocephalus with increase of intracranial tension, a choked disk can scarcely occur.

A second point of interest concerns the possible relation of the willisian vessels to the production of field defects. This possibility has been ably and thoroughly discussed from a theoretical standpoint by de Schweinitz [8] in his Bowman Lecture some years ago, though, to be

8. De Schweinitz, G. E.: Concerning Certain Ocular Aspects of Pituitary Body Disorders, Mainly Exclusive of the Usual Central and Peripheral Hemianopic Field Defects, Tr. Ophth. Soc. **43**:12-109, 1923.

sure, at this time one had scarcely begun to distinguish between suprasellar and intrasellar lesions. The dissections, 125 in number, that were made by Schaeffer,[9] which furnished the basis of de Schweinitz' conclusions, are important in that they show the chiasm from above as it is surgically brought to view, whereas the usual pictures of the region are drawn from below after the removal of the brain in its customary fashion, which gives a misleading idea of the regional topography.

De Schweinitz and Schaeffer showed (fig. 45) clearly that the chiasm normally has great variations in type, from the prefixed position as illustrated in the case just described to a postfixed position in which there is abundant room for a suprasellar growth to lie exposed to view. There can be no question, however, that even a prefixed chiasm is capable of slow distension and backward displacement from the pressure

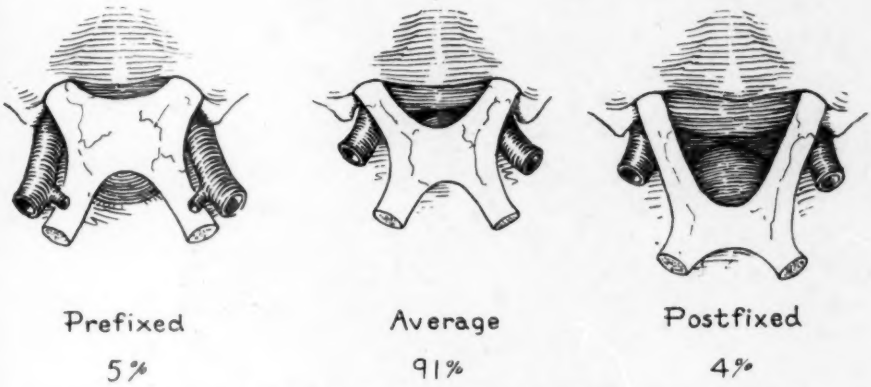

Fig. 45.—To show the three principal types of chiasm (after Schaeffer).

of such a tumor as a suprasellar meningioma which arises within its anterior embrace.

Similarly the evident prefixed position, found in the case just reported (fig. 44), may have been less anatomic than due to the pressure from behind of the largely retrochiasmal lesion. And in like fashion, an extreme dilatation of the third ventricle from obstructive hydrocephalus may so crowd the chiasm forward against the chiasmatic sulcus as to cause extreme pressure atrophy of the crossed bundles (fig. 46). Only under pathologic circumstances of this sort have I ever seen a chiasm actually in contact with the sulcus chiasmatis, where it was commonly suposed to lie until the studies of Schaeffer emphatically showed the incorrectness of this assumption.

9. Schaeffer, J. P.: Some Points in the Regional Anatomy of the Optic Pathway With Especial Reference to Tumors of the Hypophysis Cerebri and Resulting Ocular Changes, Anat. Rec. **28**:243-279, 1925.

All these matters deserve further study now that the region is coming to be so frequently exposed at operation. It is conceivable that the prechiasmal or postchiasmal position of a median suprasellar growth may come to be determinable by the character of the field defects, for if the position of the crossed macular bundle lies, as is claimed, in the posterior part of the crossing, a macular scotoma may be more likely to precede the loss of vision in the outer temporal fields in a posteriorly situated lesion than in one anteriorly situated.

When a tumor such as a craniopharyngioma is overlain by a thinned and widened chiasm, how far the surgeon is entitled to go in exposing it is a matter that must await further experience. In view of the ultimate outcome of the case just reported (case 8), I had regrets that the chiasm was not transected, for the lesion might conceivably have

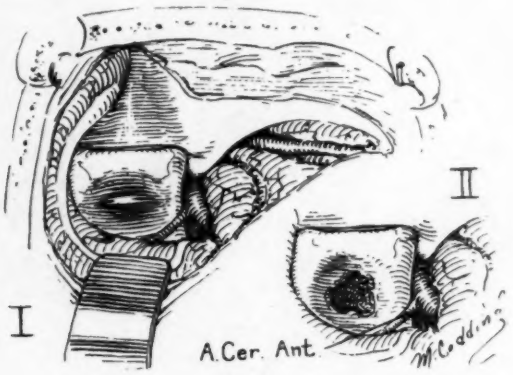

Fig. 46.—Showing the rare position of the optic chiasm actually in apposition to the sulcus chiasmatis owing to its having been crowded forward by a bulging third ventricle in a case of chronic hydrocephalus.

been sufficiently excavated to lessen the likelihood of a returning hydrocephalus. This measure, as described in case 7, has now been resorted to in five instances, all of the patients already having a bitemporal hemianopsia and a prefixed chiasm which wholly concealed the lesion. Ready access was had to the tumor, and at least a partial extirpation of the growth was thereby made possible. In all but one instance, however, they were large, more or less calcified lesions with an advanced syndrome, and only once was a suprasellar cystic tumor of the type described in case 6 encountered. This case would have been a more suitable one for the present report, as the chiasmal syndrome was uncomplicated by other secondary symptoms, but the patient was only 15 years of age, which practically excluded the diagnosis of meningioma. The essentials are as follows:

A normally adolescent girl was admitted to the hospital with symptoms of rapid and bilateral failure of vision of only six weeks' duration, associated with

frontal headaches and interruption of menses. There was a slight optic pallor of yellowish tinge with lower bitemporal field defects and large scotomas leaving only 20/200 vision in each eye. The pituitary fossa was of normal configuration, but the roentgenograms disclosed a minute intrasellar calcified fleck.

At operation, under local anesthesia, a small cyst precisely similar to that shown in figure 35 was merely tapped, and 2 cc. of discolored fluid with cholesterin crystals was sucked out. No attempt was made to dislodge the cyst. Normal visual acuity and normal field peripheries were almost immediately regained.

Within a month after her discharge, the former symptoms had wholly reappeared, and she was hurriedly brought back to the hospital. The fields at this time showed (fig. 47) almost precisely the same defects as at the previous admission, with a "breaking through" of the huge lower temporal scotoma on the left

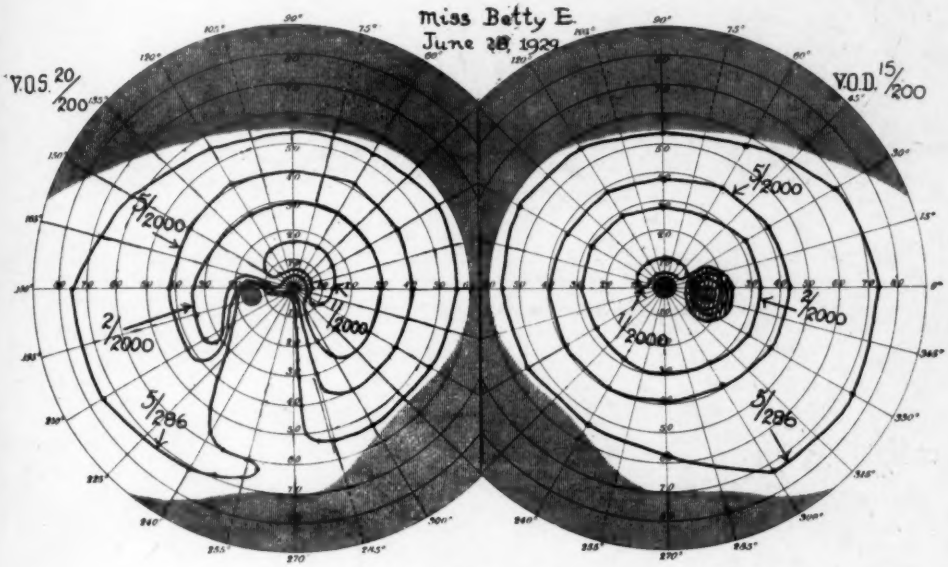

Fig. 47.—Visual fields (screen and perimeter) from a case of recurrent suprasellar craniopharyngioma in a girl of 15 years. Note the enlarged blind spot and central scotoma on the right and what was probably a similar condition on the left that has extended below and broken through all peripheries (cf. fig. 50).

and an enlarged blind spot with central scotoma on the right. Vision in the left eye was 20/200; in the right eye 15/200. A further rapid loss of vision occurred in the right eye, so that within three days the blind spot and macular scotoma had fused, and the huge scotoma had spread over the lower temporal field (fig. 48), though it had not as yet actually "broken through." Vision in the right eye was 8/200.

At a second operation, again under local anesthesia, the chiasm was transected, and the cyst was in large part if not wholly dislodged. There was again immediate improvement in visual acuity with 20/15 vision in the left eye and normal 20/25 vision in the right eye. There was of course a permanent sharply cut bitemporal hemianopsia (fig. 49) which interfered almost not at all with reading.

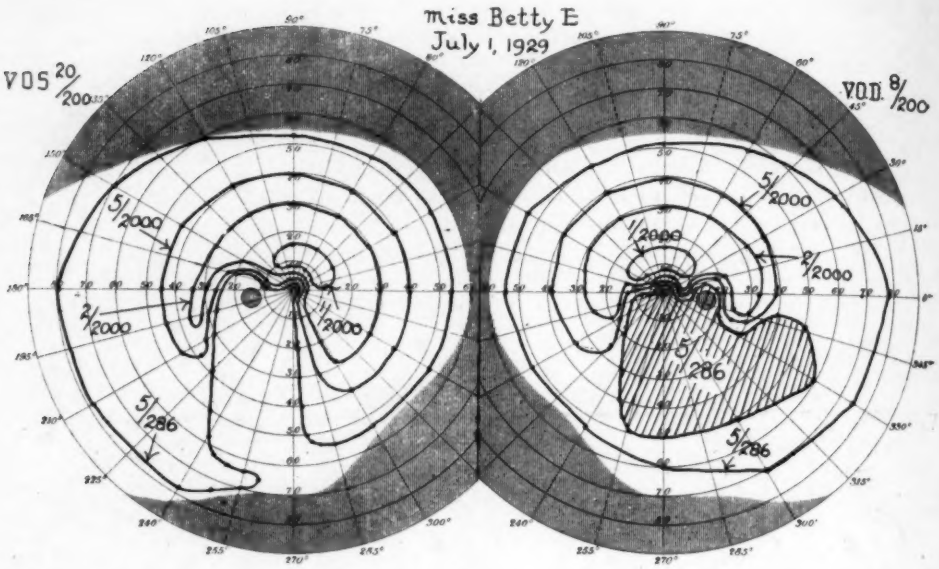

Fig. 48.—(Cf. fig. 47.) Note, in the right eye, the rapid expansion downward of a large scotoma which probably, as on the left, would have broken through the outer perimetric field in a few days more.

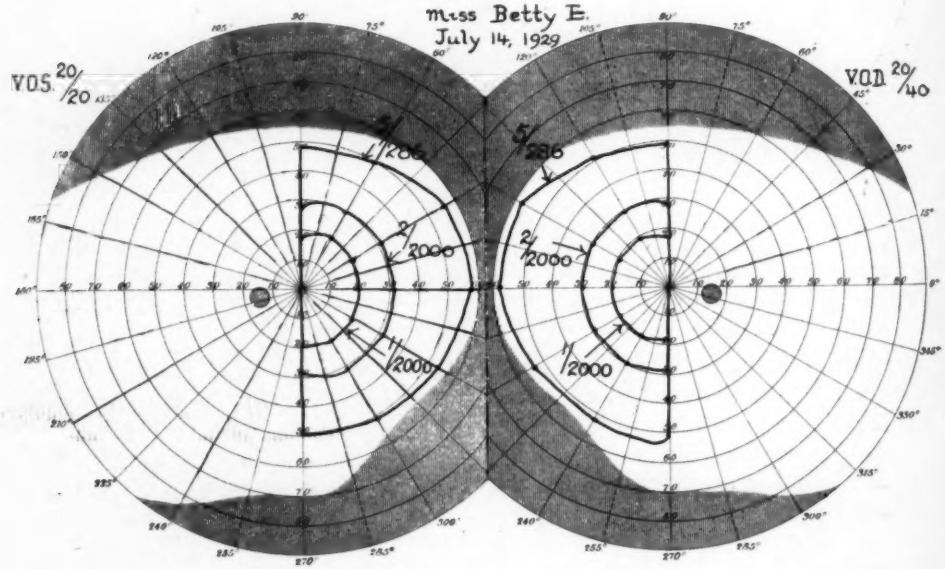

Fig. 49.—Fields of same case as that illustrated in figures 47 and 48 after transection of chiasm in order to expose and remove small cystic tumor. Note cleancut bitemporal hemianopsia bisecting macula with marked improvement in visual acuity in both eyes (eight days after operation).

Should the enucleation of similarly concealed tumors thus prove to be possible, it may encourage earlier operation in some of these cases, for a permanent bitemporal hemianopsia is a small price to pay to gain release from the other far more serious symptoms caused by these craniopharyngiomas in the course of their progressive enlargement. The problem is much simpler in the many tumors of like nature that lie freely exposed between the widened anterior crotch of a postfixed chiasm, but their consideration lies outside the scope of this paper.

(To be Continued)

ACUTE TUBERCULOUS PERIPHLEBITIS OF THE RETINA AND OPTIC NERVE *

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AND
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For many years the choroidal tubercle has been designated as the most conspicuous ocular sign in the course of acute generalized miliary tuberculosis. Tubercles of the conjunctiva have been seen but rarely, while fresh tubercles of the iris arising in the acute course of the disease are a curiosity. Gilbert [1] reported such a case and stressed the distinction of such lesions from the so-called miliary tubercles found on the iris in chronic ocular tuberculosis. For these he reserved the term "tuberculids." Tubercles of the retina in acute generalized miliary tuberculosis are even still rarer. Professor Fuchs,[2] in discussing primary retinal tuberculosis, stated that the lesion was described but once, by Litten;[3] further, he said that small hemorrhages in cases of choroidal miliary tuberculosis should not, without other proof, be taken to indicate tubercle of the retina.

Investigation of the contemporary literature reveals that the lesion was in reality described by Perls [4] in 1873, and the facts of his case are so remarkably similar to those in the one to be reported as to bear repetition:

A six months' old child was noted to develop a cloudy cornea in one eye. There were fresh nodular lesions on the surface of the iris. Six weeks later, the child was seized with an acute infective process and succumbed in a few days. At autopsy, miliary tubercles were found in various organs, although the meninges were free. In the eye, the iris contained caseating tubercles. None were found in the choroid, but in the retina were a number of prominent submiliary sharply circumscribed nodules, which grossly and on section were seen to be situated along the veins. Otherwise tubercles could not be differentiated in the retinal tissue.

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* Submitted for publication, Feb. 21, 1930.

* Read before the New York Academy of Medicine, Section of Ophthalmology, Feb. 17, 1930.

* From the Laboratories and Ophthalmological Division of the Mt. Sinai Hospital.

1. Gilbert, in Graefe-Saemisch: Handbuch der gesamten Augenheilkunde, ed. 11, Leipzig, Wilhelm Engelmann, part 2, vol. 5, chap. 2, p. 177.

2. Fuchs: Ueber primäre Tuberkulose der Netzhaut, Arch. f. Ophth. **101**:14, 1919.

3. Litten: Ueber acute Miliartuberculose, Samml. klin. Vortr., 1877, no. 119, p. 1061.

4. Perls: Zur Kenntnis der Tuberculose des Auges, Arch. f. Ophth. **19**:221, 1873.

Perls' description of the lesions, together with the illustrations, leaves little doubt that this was an instance (and incidentally the first) of periphlebitis tuberculosa retinae and not, as indicated by some authors, tubercles of the retinal tissue proper. In the light of later studies, this case furnished an excellent clue to the origin of the periphlebitic disease; namely, a previous infection in the anterior portion of the uvea. Considered from this standpoint, Perls' case appears to have been totally disregarded in the literature, since the conclusions reached as to the relation between the periphlebitic lesion in the retina and anterior disease were based on an entirely different type of tuberculous infection in the eye.

Periphlebitis retinae forms an interesting chapter in ocular tuberculosis. Fleischer,[5] in 1914, confirmed pathologically Axenfeld's[6] earlier clinical impression regarding the tuberculous nature of the disease causing recurrent vitreous hemorrhages and proliferating changes in the retina. In the presence of intact choroid and a single focus in the ciliary body, Fleischer found hemorrhages along the veins and on the surface of the retina, while the veins showed tuberculous nodules in their walls and were even occluded by endophlebitis. Gilbert[7] had spoken of phlebitis and mesophlebitis as well in his cases of diseases of the blood vessels in the retina. Numerous reports have confirmed both the nature of the lesion and the views regarding the source of the hemorrhages, especially in the young. Meller,[8] in 1922, stressed that the pathway of infection of the retina complicating tuberculous iridocyclitis is by way of the perivascular lymph spaces, to produce periphlebitis, and in some instances affecting the nerve itself in sequence, causing retrobulbar neuritis. This view has been generally accepted with respect to the periphlebitic lesion, although it is interesting to note its occurrence as well in severe necrotic lesions of the choroid and retina (Bergmeister[9]), in overwhelming metastatic infections (tuberculous panophthalmitis) such as those described recently by Schöpfer[10]

5. Fleischer: Die juvenile Periphlebitis mit ihren Folgeerscheinungen, Klin. Monatsbl. f. Augenh. **52**:769, 1914.

6. Axenfeld: Ueber die Bedeutung der Tuberkulose in der Ätiologie der intraokulare Hämorrhagien, Klin. Monatsbl. f. Augenh. **49**:28, 1911.

7. Gilbert: Ueber juvenile Gefässkrankungen des Auges, Arch. f. Augenh. **75**:1, 1913.

8. Meller: Ueber die Mitbeteilung der Netzhaut an der Iridocyclitis, Ztschr. f. Augenh. **47**:247, 1922.

9. Bergmeister: Ueber die nekrotisierende intraokuläre Tuberculose, Ztschr. f. Augenh. **53**:175, 1924.

10. Schöpfer: Tuberculosen metastatischen Ophthalmie, Klin. Monatsbl. f. Augenh. **83**:31, 1929.

and in Verhoeff's [11] case of localized chorioretinitis. A notable instance of spread of infection along perivascular channels is that of Frank,[12] in acute tuberculous ophthalmia in a boy, aged 8, with typical periphlebitic nodules. There were no hemorrhages in this case. In fact most authors stressed the long-standing disease in the retina before the occurrence of hemorrhage.

The case to be reported, which has only one to parallel it, that of Perls, represents the most extensive involvement of the globe as yet reported in the actual course of acute generalized miliary tuberculosis. Besides the features of Perls' case, it includes tubercles of the choroid, papilla and nerve, and, in addition, retinal hemorrhage, a complication identified hitherto with periphlebitis tuberculosa only as it occurred in the chronic form as described by Fleischer. It illustrates as well the dependence of the posterior lesion on a previously acquired iridocyclitis.

REPORT OF CASE

History.—A full term Porto Rican boy had been well until the age of 6 months, when the pupil of the right eye became distorted and the eye grew cloudy. The child received no treatment. Two months later he started to vomit and became restless, and the day before admission to Dr. Béla Schick's service he went into a convulsion.

Examination.—The right pupil was noted to be irregular; its margins were covered with fresh exudate binding them to the lens. The whole iris was vascularized, especially in its nasal half. Above and nasally were large newly formed vessels. Scattered over the iris were round, fluffy, grayish elevations, some of which coalesced. The lens was covered by exudate. The left eye was normal. The left fundus was negative for abnormality; the right fundus could not be seen. Death occurred the day after admission, on the eighth day of the acute illness. The clinical diagnosis was acute miliary tuberculosis and tuberculous meningitis. The lesion of the eye was taken to be acute tuberculous iridocyclitis with tubercles of the iris.

Autopsy.—Autopsy showed: acute miliary tuberculosis of the lungs, liver, spleen, kidneys, intestines and skin. There were also tuberculous meningitis and tuberculous mesenteric lymphadenitis.

Microscopic Ocular Structure.—The anterior chamber contained an eosin-staining material (probably serum) which extended to the ciliary region and occupied a considerable portion of the posterior chamber. Portions of Descemet's membrane and a few corneal fibers were dissolved. Pigment granules and lymphocytes had wandered into these defects. Similar deposits covered Descemet's membrane where the latter was intact. The body and base of the iris were adherent to the cornea, obliterating the angles. The fusiform tubercles were nearer the base and bulged into the cornea. The middle third, which was filled with lymphocytes, was intact; the pupillary zone was the seat of marked necrosis and con-

11. Verhoeff, F. H.: Histologic Observations in a Case of Localized Tuberculous Chorioretinitis, Arch. Ophth. **1**:63 (Jan.) 1929.

12. Frank: Zur Kenntnis der perivasculären Tuberkulosenverbreitung im Auge, Ztschr. f. Augenh. **57**:301, 1923.

tained new capillaries which broke through the surface into the mass of exudate covering the iris. The pupillary edge was adherent to the lens capsule by a mass of tissue consisting of lymphocytes, epithelioid and giant cells of the Langhans type. The posterior iris epithelium was exfoliated to a marked degree.

There was tubercle formation in the meshes of the suspensory ligament and in the perilenticular space. Most of the iris and ciliary retinal epithelium was drawn into the mass. Scattered in this tissue were disintegrated pigment, fibrin and young connective tissue cells. The ciliary muscle was only slightly infiltrated, but the anterior portion of the ciliary body and its processes were replaced by

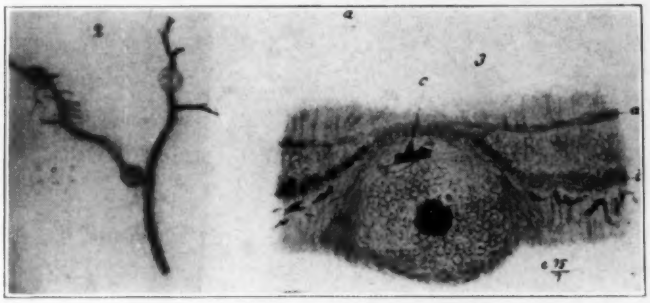

Fig. 1.—Periphlebitic tubercles (gross and microscopic) in Perls' case of acute miliary tuberculosis (Graefe's Arch. f. Ophth., 1873, vol. 19).

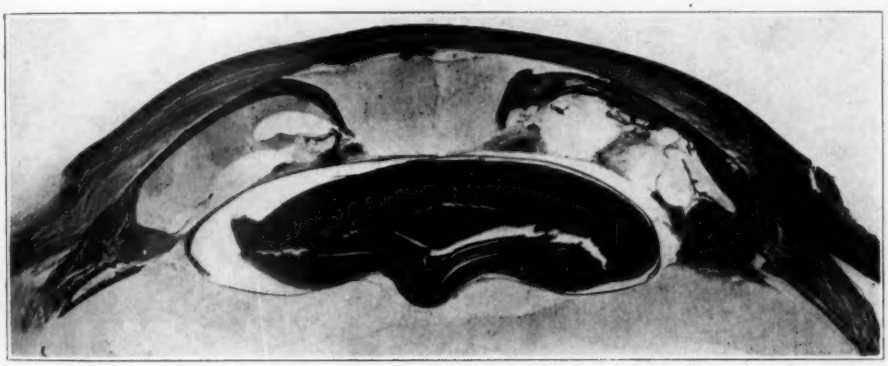

Fig. 2.—Anterior segment showing acute tuberculous iridocyclitis, with tubercle of the iris and ciliary body, and exudate binding pupillary edges to the lens capsule.

tuberculous granulation tissue. The pars plana bore tuberculous tissue on its surface, while the adjacent orra serrata contained a few lymphocytes which may have been derived from the infiltrated vitreous.

The lens was intact.

The sclera adjacent to the ciliary body showed a few round cells.

The choroid contained a few tubercles.

For the most part, the retina was covered with serum and thrown into folds. Its limiting membrane here and there bulged with crescents of fibrin, a few lymphocytes and red blood cells. Near the disk were large masses of blood and

fibrin lying free on the retina outside the limiting membrane. Toward the periphery the small veins were collared with round cells. In serial section, these veins were occluded in some portions of their course, or else were replaced by tuberculous granulation tissue. Except for small collections of lymphocytes jutting into the vitreous (such as described by Verhoeff in his case of localized chorioretinitis), the retinal tissue was free from tubercle. Figure 4 illustrates the disposition of

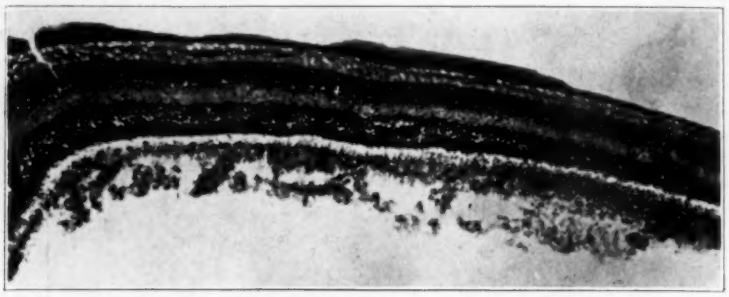

Fig. 3.—Blood and fibrin on the surface of the retina near the disk.

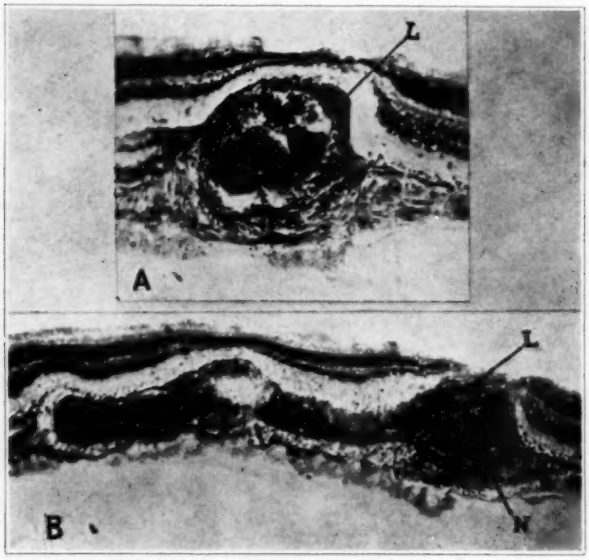

Fig. 4.—In *A*, the lumen, *L*, is displaced upward by the tubercle in the wall; *B* shows the same vein obliterated by the nodule, *N*, and its distal portion dilated with blood. There is blood on the surface of the retina.

tubercles along the larger veins and the most probable mode of origin of the bleeding. The large giant cells appeared at first glance to occupy the lumen of the vessel. Closer study showed the tubercle mass to occupy the wall of the vein and the perivascular space, displacing the lumen and what remained of its wall upward. The granular layer had undergone pressure atrophy, while the nuclear

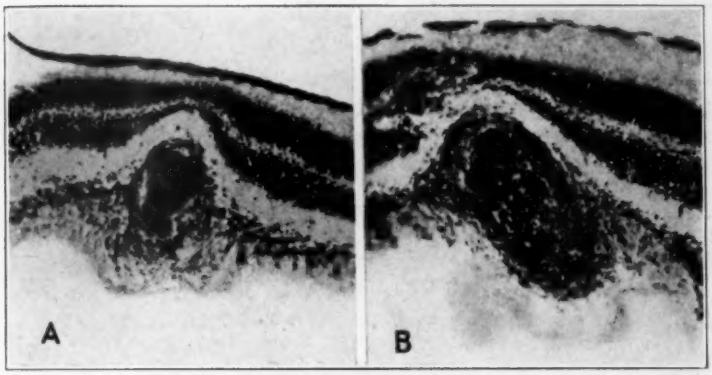

Fig. 5.—Sections of the same vein as in figure 4. *A* shows a giant cell apparently in the lumen, also periphlebitis; *B*, phlebitis as well, and possibly also obliterating endophlebitis.

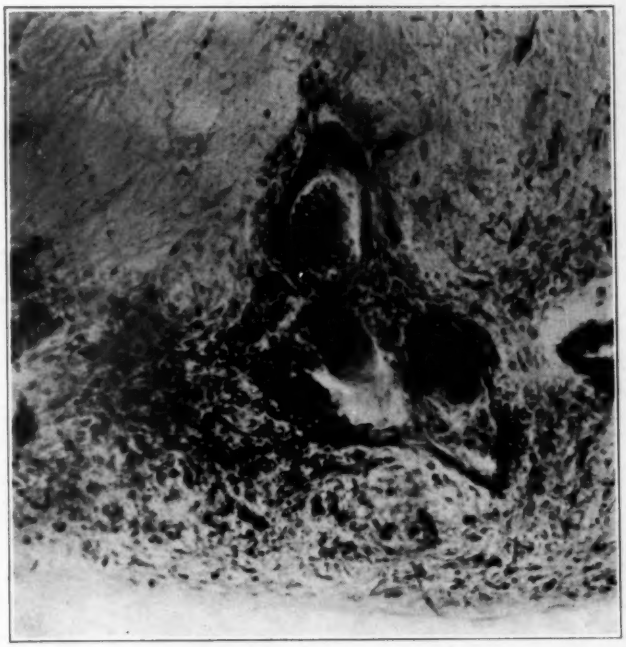

Fig. 6.—Tuberculous infiltration surrounds the central vessels and extends into the disk. Two sections of the vein below contain giant cells; the artery above contains blood.

layers were reduced to the merest thinness. On further section (fig. 4 *B*), the distal portion of the vein was greatly dilated with blood, while the ganglion and fiber layers were filled with fibrin, and the surface of the retina bore extravasated blood cells. The effect was therefore pressure occlusion; the bleeding was most likely due to stasis.

In other portions of the retina, it was difficult to ascertain the exact relation of the tubercle to the wall and the lumen of the vein. For example, figure 5 *A* shows a giant cell seemingly in the lumen, denoting possibly extension of the infection through the wall into the intima. Figure 5 *B*, at a slightly different level, shows apparently only a mass of tuberculous granulation tissue, so that it is hardly possible to tell whether one is dealing with a massive periphlebitic and phlebitic nodule obliterating the lumen or actual occlusion due to proliferation of tuberculous granulation tissue in the intima. Although the invasion of the latter could not be excluded, one is inclined to favor occlusion due to compression from without rather than from an inflammatory process within the lumen.

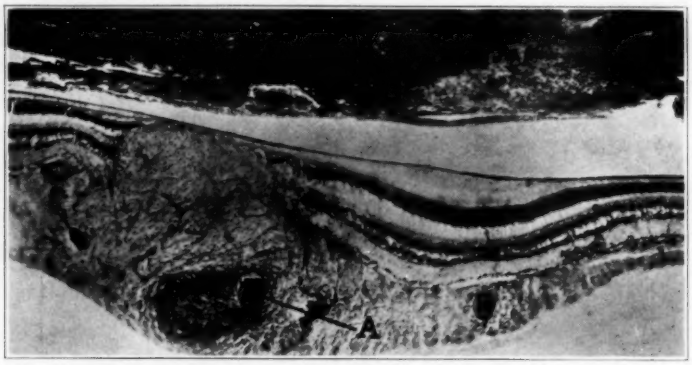

Fig. 7.—The central artery, *A*, in the disk is intact. The vein is replaced by tubercle tissue. Above and to the right is a choroidal tubercle.

Sections stained for tubercle bacilli failed to show the organism in the nodules about the veins.

The relation of vein and tubercle was more definite in the disk. In figure 6 are two sections of the central vein and one of the central artery above. It is clear that the giant cells occupy the lumen of both veins. About the entire group, including the artery, were lymphocytes and epithelioid cells, while the nasal portion of the disk was similarly infiltrated. Study of further sections showed a gradual occlusion of the vein, so that it was replaced entirely by tubercle (fig. 7). Evidently tubercle had broken through into the intima. A mass of tuberculous granulation tissue extended along the vein in longitudinal section from the pit to a point some distance behind the lamina cribrosa, but the process was confined to the sheath of the vessel, as there was no actual invasion of the nerve in either its intra-ocular or its retrobulbar portion.

COMMENT

It is generally agreed that miliary tuberculosis of the choroid occurs by way of the blood stream. The fact that this process ordinarily

leaves the retina and iris uninvolved points strongly to the dependence of the retinal periphlebitic lesion on the recently acquired iridocyclitis.

In estimating the comparative age of anterior and posterior lesions, there is some evidence that the retinal lesion, and more especially the bleeding, occurred with the onset of the acute generalized process. The presence of young fibroblasts and new capillaries in the anterior chamber indicates an attempt at healing of the primary lesion of the iris and ciliary body. No such attempt at organization is seen in the retina, and there is no evidence of proliferative change or cicatrization of the hemorrhagic areas on the retina. Very likely, with the advent of an overwhelming infection of the blood stream with tubercle bacilli, there was a relighting of the disease in the iris and a pouring out of exudate into the vitreous. Periphlebitis could then be traced to direct infection of the retina or, more specifically, of the flat portion of the ciliary body and the adjacent orra serrata.

The extreme difficulty with which the retina is infected with tubercle bacilli by way of the blood stream is seen in the attempts to produce tuberculosis in the eye experimentally. Finnoff [13] observed the lesion independent of that in the choroid only twice in a series of forty-six animals infected with tubercle bacilli by way of the carotid artery. These lesions are necrotic and correspond to the severe necrotizing lesions of metastatic origin in the case of chronic pulmonary tuberculosis described by Bergmeister,[9] in which bacilli were found in the lesion, and in which the route of infection was directly hematogenous.

13. Finnoff, W. C.: Changes in Rabbits' Eyes Following Injection of Living Bacilli into Common Carotid Artery, Am. J. Ophth. **7**:81, 1924.

THE PATHOGENESIS OF ACUTE GLAUCOMA

I. CLINICAL AND PATHOLOGIC STUDY *

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The present study represents an attempt to establish a working hypothesis in regard to the nature of acute glaucoma. Hitherto, in the elaboration of theories of glaucoma, both acute and chronic glaucoma have been considered together. This effort at a unitary theory of glaucoma perhaps has come about because the end-results of acute and chronic glaucoma present many points of identity. It may even be possible that at some future time the same ultimate cause of both diseases will be discovered. Such an ultimate cause, however, is beyond the scope of the present study. But whatever the ultimate cause or causes of acute and chronic glaucoma, there can be little doubt that the mechanism by which an attack of acute glaucoma develops is essentially different from that which produces the clinical picture of chronic simple glaucoma. We are therefore justified in omitting the problem of chronic glaucoma from consideration when we ask the question: "How does an attack of acute glaucoma develop?" In what way must the normal physiologic mechanism regulating intra-ocular pressure be upset in order to initiate an attack of acute glaucoma?

My attention was first directed toward this problem by a study of the cases of acute glaucoma that are complicated by necrosis of the iris. This condition was first described by Fuchs and has since been observed by various authors. Necrosis of the iris is not uncommon in cases of acute glaucoma with extremely high tension (fig. 1). Generally a circular zone of iris extending outward from the pupillary margin is affected. This circular distribution clearly indicates that a local disturbance in the vascular system of the iris is not responsible for the lesion, for each radial artery in the iris supplies a wedge-shaped zone, and the circular vessels are furnished with abundant anastomoses. The necrosis must, then, be a direct result of a pressure sufficient to cause collapse of the capillaries supplying the region affected. One must therefore conclude that, in these cases, the intra-ocular pressure exceeds the pressure in the capillaries of the pupillary portion of the iris.

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* From the Wilmer Ophthalmological Institute of the Johns Hopkins University and Hospital.

Studies in general physiology by Starling,[1] Van Slyke,[2] Laurence Henderson [3] and others, applied to ocular physiology by Hertel,[4] Duke-Elder [5] and others, conclusively prove that under normal conditions, on the average, the intra-ocular capillary pressure exceeds the intra-ocular pressure by the osmotic pressure of the plasma proteins, approximately 25 mm. of mercury. This difference is maintained by virtue of the fact that the capillary walls are normally impermeable to the protein molecules of the blood plasma. Applying these considerations to cases of acute glaucoma with necrosis of the iris, one is forced to conclude that if the osmotic barrier of the capillaries throughout the eye remains unchanged, the pressure in some intra-ocular capillaries must exceed that of the iris capillaries by upward of 25 mm. of mercury. Manifestly, the number of high pressure capillaries must be great, and

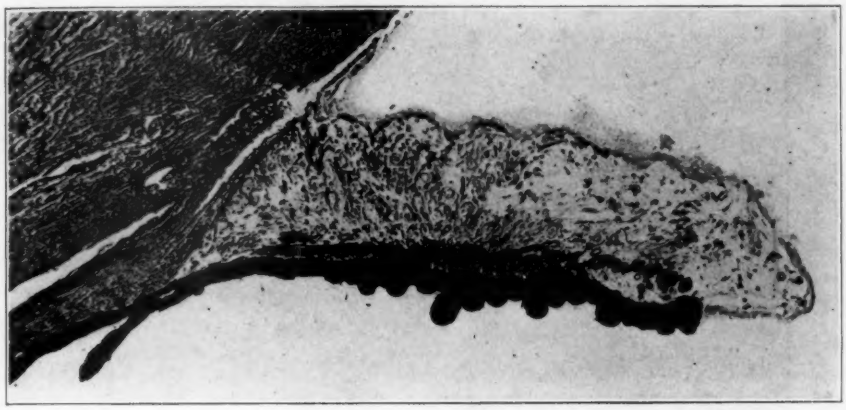

Fig. 1 (case 10).—Necrosis of the pupillary margin of the iris.

the excess of their pressure over that required for equilibrium with the intra-ocular pressure must be considerable if the extremely high intra-ocular pressure in these cases is to be maintained. But the intra-ocular capillaries are all supplied by branches of the same artery, and while extreme differences in pressure may occur between one capillary and the next, it seems unlikely that the average capillary pressure in one

1. Starling, E. H.: J. Physiol. **14**:312, 1895-1896.

2. Van Slyke, D. D.: Factors Affecting the Distribution of Electrolytes, Water and Gases in the Animal Body, Philadelphia, J. B. Lippincott & Company, 1926.

3. Henderson, Laurence J.: Blood, New Haven, Conn., Yale University Press, 1928.

4. Hertel, E.: Deutsche Ophth. Gesellsch. **42**:73, 1919.

5. Duke-Elder, W. S.: Brit. J. Ophth., Monograph Supplement III, London, George Pulman and Sons, Ltd., 1927.

region in the eye should exceed that in another region by anything approaching 25 mm. of mercury.

One reaches what appears to be a reductio ad absurdum, but an alternative view is possible: If in any extensive bed of intra-ocular capillaries the osmotic barrier against the plasma proteins were destroyed, these capillaries could maintain the high level of intra-ocular pressure even though their pressure exceeded the intra-ocular pressure by only a few millimeters. As to the probable site of such a capillary lesion the ciliary body seems most likely. Edema of the ciliary body in acute glaucoma and turgidity of the ciliary processes

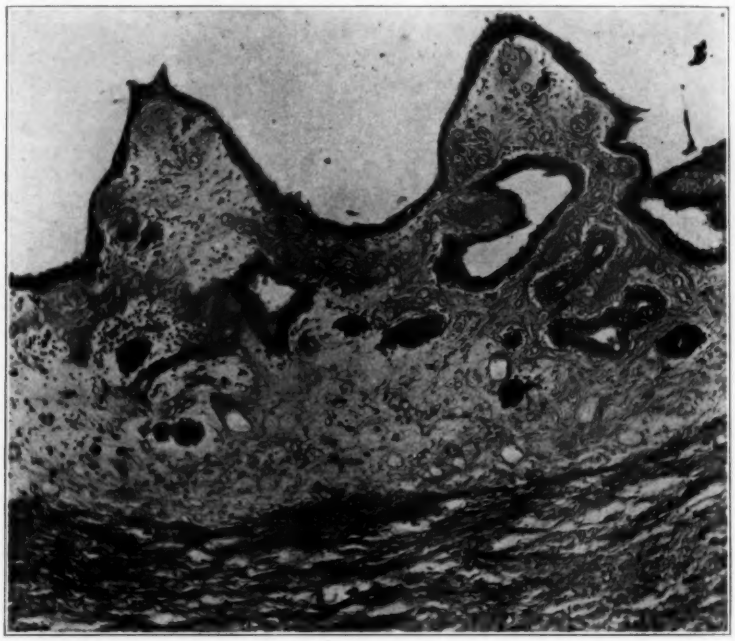

Fig. 2 (case 1).—Cross-section through the anterior portion of the ciliary body, showing edema of the ciliary processes with albuminous exudate about the capillaries.

have long been known to be characteristic of acute glaucoma. Shallowness of the anterior chamber and obstruction of the filtration angle in acute glaucoma have generally been explained as a result of the swelling of the ciliary body and processes. It is natural, therefore, to search for the evidences of capillary damage in this region. With this introduction, I pass on to the report of the detailed examination in serial section of the ciliary region of eleven eyes affected with acute glaucoma. These cases have not been selected out of a larger number, but represent all the cases of undoubted acute glaucoma in which I have a

histologic study, excluding only those in which the pathologic picture was markedly altered as the result of operative intervention. Cases 1, 2, 3, 4 and 5 illustrate varying degrees and durations of relatively uncomplicated acute glaucoma. In cases 6, 7 and 8 the glaucoma was secondary to thrombosis of the central retinal vein. Cases 9, 10 and 11 represent mild instances of secondary glaucoma complicated by an acute rise of intra-ocular tension.

REPORT OF CASES

CASE 1.—Twenty years before the present observations a foreign body in the eye was extracted with a magnet. One year before, a mature cataract was found,

Fig. 3 (case 1).—Same region stained by Goodpasture's method for fibrin, which is seen in black strands surrounding the capillaries.

and an attack of acute glaucoma occurred, with recovery under the use of miotics. Two days after another severe attack of acute glaucoma, enucleation was performed.

Pathologic Observations.—There was a shallow anterior chamber, and marked edema of the ciliary body. The root of the iris was pushed forward and obstructed the filtration angle. No atrophy of the iris or the ciliary body and no organized peripheral anterior synechia were found. The ciliary processes were swollen, thickened and filled with a homogenous coagulum. In the anterior tips of the ciliary processes this coagulum showed definite concentric lamellae about the capillaries and had the characteristic staining reaction of fibrin. There was slight atrophy of the retina and optic nerve, with shallow excavation of the optic disk (figs. 2 and 3).

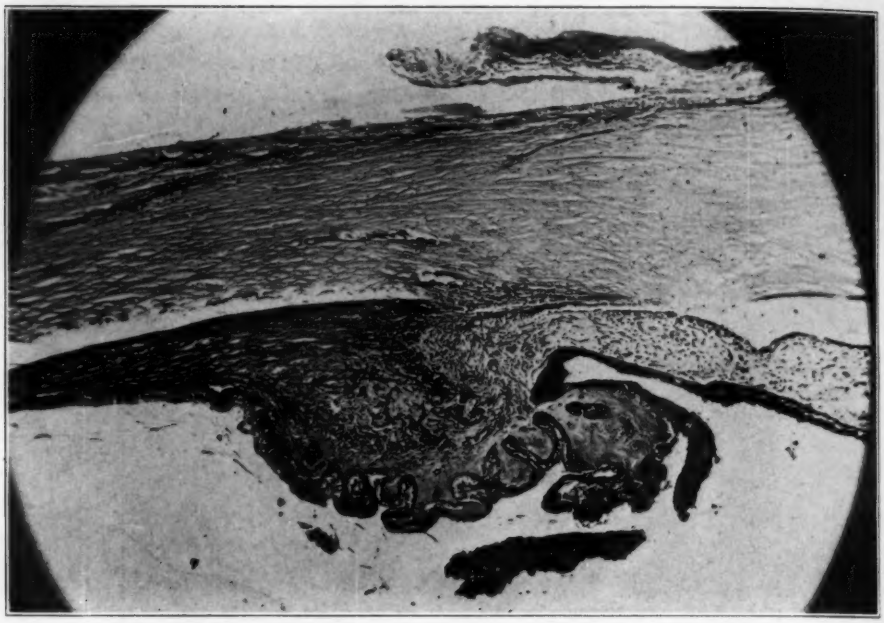

Fig. 4 (case 2).—Edema of the ciliary body and processes pushing the root of the iris against the cornea.

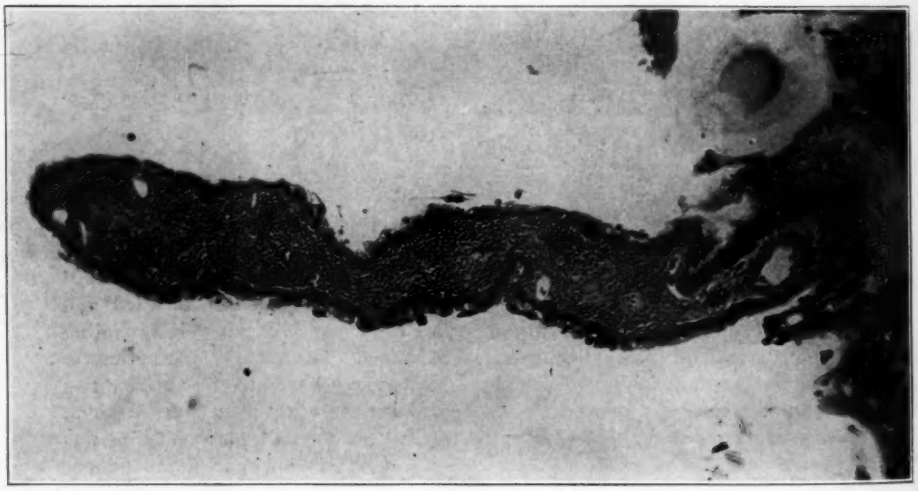

Fig. 5 (case 2).—Hemorrhage in the ciliary process.

CASE 2.—Absolute glaucoma followed acute glaucoma.

Pathologic Observations.—There was edema of the ciliary body. The root of the iris was displaced forward but was not adherent to the cornea. Necrosis of the pupillary margin of the iris was seen. There was marked swelling of the ciliary processes, especially in their anterior portions, with serum and fibrin about the capillaries. Hemorrhages were found in some ciliary processes. Slight atrophy of the retina and of the optic nerve, and shallow excavation of the optic disk were observed (figs. 4, 5 and 6).

CASE 3.—Acute glaucoma began two months before the present observations. Posterior sclerotomy, performed one month after the onset of the condition, relieved tension for three weeks. There was a recurrence of acute glaucoma, and one week later enucleation was performed.

Fig. 6 (case 2).—Hyalinized exudate in the ciliary process.

Pathologic Observations.—There were moderate atrophy and marked edema of the ciliary body. A shallow anterior chamber and an organized peripheral anterior synechia with atrophy of the iris were observed. The ciliary processes were swollen and thickened; they contained serum and fibrin, especially in their anterior tips. Advanced atrophy of the retina and of the optic nerve was seen, as well as excavation of the optic disk (fig. 7).

CASE 4.—Cataract developed four years before the present observations. Acute glaucoma developed, and four weeks later enucleation was performed.

Pathologic Observations.—Examination revealed bulous keratitis, a shallow anterior chamber and anterior dislocation of the root of the iris, without adhesions to the cornea, a hypermature cataract and marked edema of the anterior portion of the ciliary body. Serum and fibrin were found in the anterior portions of the ciliary processes. Desquamation of the epithelium of the ciliary processes had occurred in some places. There was slight atrophy of the retina and the optic nerve, with shallow excavation of the optic disk (fig. 8).

CASE 5.—The patient had had recurrent attacks of acute glaucoma of increasing severity for two years. Absolute glaucoma developed, and enucleation was performed.

Pathologic Observations.—The root of the iris was edematous, thickened and adherent to the cornea. The pupillary portion of the iris, on the other hand, was thin and atrophic. Other observations were: atrophy of the ciliary body, with marked edema; engorgement of the ciliary processes, which were thickened and contained a coagulum more dense and hyalin-like than that in the previous cases; scattered retinal hemorrhages; atrophy of the retina and of the optic nerve; excavation of the optic disk, and engorgement of the choroid, with thrombosis of one choroidal vein behind the equator.

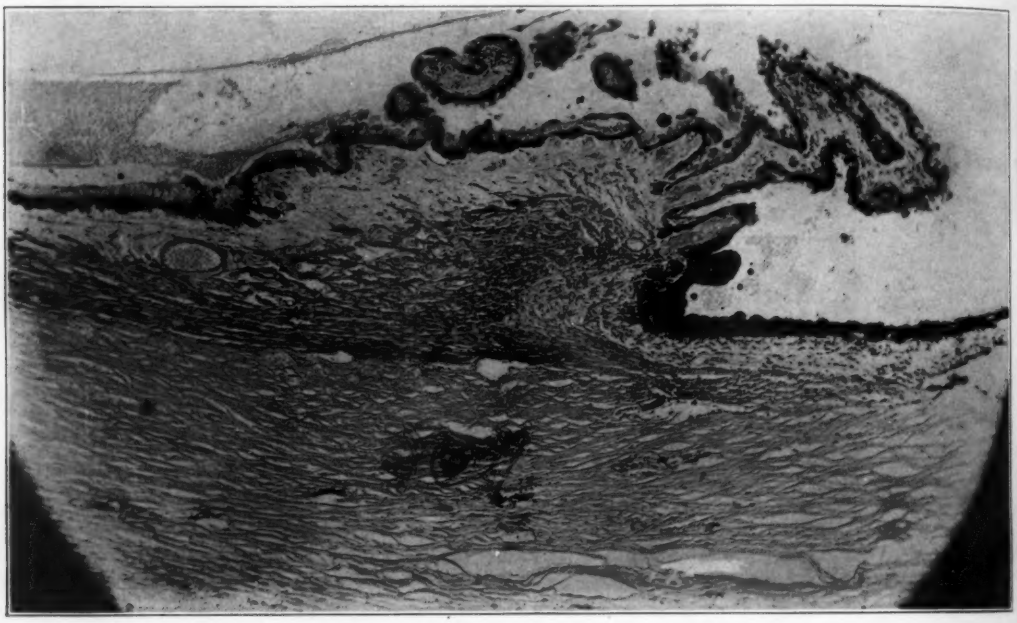

Fig. 7 (case 3).—Edema of the ciliary body.

CASE 6.—Thrombosis of the central retinal vein developed. Two months later, and three weeks after the onset of acute glaucoma, enucleation was performed.

Pathologic Observations.—Edema of the ciliary body, a shallow anterior chamber and peripheral anterior synechia with beginning organization were seen. The ciliary processes were swollen and thickened, and contained serum and fibrin. There were numerous retinal hemorrhages. Collateral venous channels were seen near the optic disk. Partial optic atrophy had occurred, with shallow excavation of the optic disk.

CASE 7.—Thrombosis of the central retinal vein developed three years before the present observations, and was followed, three months later, by acute glaucoma with partial recovery. There had been a recent recurrence of the glaucoma. Posterior sclerotomy and cyclodialysis were performed, without relief.

Pathologic Observations.—Examination revealed advanced atrophy of the ciliary body and the iris, beginning ectropion uveae, old organized peripheral anterior synechia and marked edema of the ciliary body. The ciliary processes were swollen and contained more densely staining coagulum than was observed in the previous cases. Other observations were: subchoroidal hemorrhage, hemorrhage in the anterior and posterior chambers (operative), advanced atrophy of the retina and of the optic nerve, retinal hemorrhages, collateral venous channels near the optic disk and excavation of the optic disk.

Case 8.—Blurred vision (thrombosis of central retinal vein?) developed in September, 1928. On Dec. 2, 1928, acute glaucoma was noted. A trephine operation was performed on December 10. The glaucoma recurred on December 18,, and enucleation was performed on January 8, 1929.

Pathologic Observations.—The trephine hole was filled with granulation tissue. There was moderate atrophy of the ciliary body and of the iris, with an organized

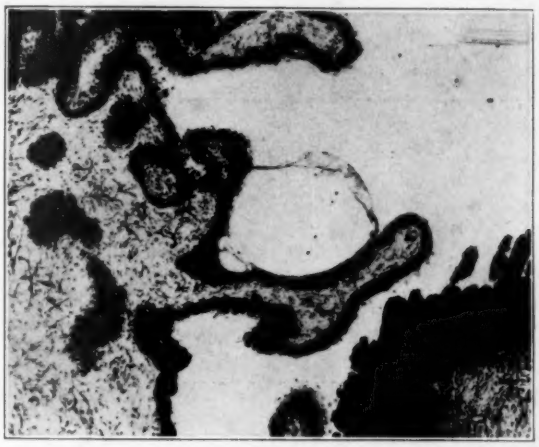

Fig. 8 (case 4).—Desquamation of the epithelium of the ciliary process.

peripheral anterior synechia and slight edema of the ciliary body. Serum and hyalin were found in the ciliary processes, not only in the anterior tips but extending back to the posterior portions. Other observations were: atrophy of the retina and of the optic nerve, numerous retinal hemorrhages, collateral venous channels near the optic disk, thrombosis of the central retinal vein and deep excavation of the optic disk.

Case 9.—The patient had recurrent iritis with secondary glaucoma. A few days after an acute exacerbation of the glaucoma, enucleation was performed.

Pathologic Observations.—Examination disclosed: bullous keratitis, a shallow anterior chamber, organized peripheral anterior synechia, an old posterior synechia but no active inflammatory reaction, marked edema of the ciliary body, marked swelling of the anterior tips of the ciliary processes, atrophy of the retina and of the optic nerve and excavation of the optic disk.

Case 10.—The eye had been injured during the patient's childhood and was amblyopic. Acute glaucoma developed, and three weeks later enucleation was performed.

Pathologic Observations.—Advanced atrophy of ciliary body and iris was present, with an old organized peripheral anterior synechia and necrosis of the pupillary margin of the iris. A fragment of the ruptured lens capsule was adherent to the posterior surface of the cornea. The shrunken lens nucleus rested on the retina in the posterior portion of the globe. Moderate edema of the ciliary body, swelling of the ciliary processes, and coagulated serum about the capillaries in the anterior portions of the ciliary processes and in the iris were observed.

CASE 11.—The patient had glaucoma. No details were obtained.

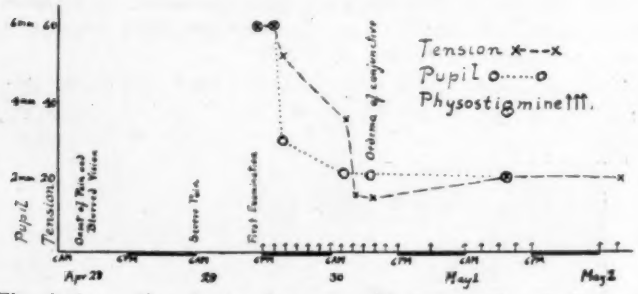

Fig. 9 (case 1).—Acute glaucoma with hypotension during recovery.

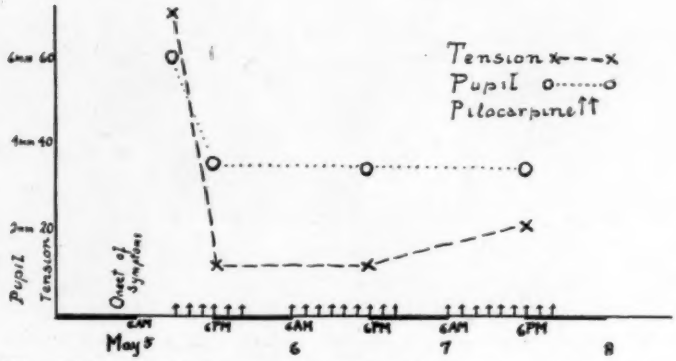

Fig. 10 (case 2).—Acute glaucoma with hypotension during recovery.

Pathologic Observations.—Examination revealed bullous keratitis with beginning ulceration; an organized peripheral anterior synechia; old adhesions between the iris and the lens; atrophy of the ciliary body, with marked edema in the anterior portions; serum and fibrin about the capillaries in the anterior portion of the ciliary processes; hemorrhagic detachment of the epithelium of one ciliary process, and atrophy of the retina and of the optic nerve without excavation of the optic disk.

COMMENT

The histologic study of the cases just reported revealed, with extraordinary uniformity, evidence of a vascular lesion in the ciliary body. In all cases the edema of the ciliary body and the ciliary processes

was sufficient to account for the shallowness of the anterior chamber and the obstruction of the filtration angle.[6] Hemorrhagic, serous or fibrinous extravasations in the ciliary processes were seen in the earlier and more acute cases, the lesion being most marked in the anterior portions of the ciliary processes, which were swollen to two or three times their normal thickness. In the later stages, the coagulum in the ciliary processes was more compact and resembled hyalin. The presence of fibrin about the capillaries indicated a leakage of plasma out of the vessels, with subsequent clotting in the perivascular spaces. The fibrin had remained in situ, while the serum had, in largest part, passed into the ocular cavity.

It seems clear, then, that acute glaucoma is associated with a vascular lesion which is localized most prominently in the capillaries of the ciliary body and processes. Such an injury to the capillary walls with hemorrhagic, serous and fibrinous extravasations could result from any one of three causes: (1) venous stasis, (2) vasomotor disturbances and (3) the presence of some irritant substance of the nature of a capillary poison in the tissues. In the present status of physiologic knowledge I can conceive of no other mechanism of capillary damage than the mechanical, nervous and chemical mechanisms suggested under these three headings.

1. Venous Stasis: The conception that venous stasis might be the cause of acute glaucoma long ago led to extensive researches. Koster [7] studied most minutely the ocular changes that followed ligation of the vortex veins. He found that during the first twenty-four hours following such a procedure certain aspects of the clinical picture of acute glaucoma developed, namely, mydriasis, shallowness of the anterior chamber and increased intra-ocular pressure. These changes, however, were only transitory. A return to normal occurred in a short time. Histologic examination of these eyes revealed massive engorgement of the ciliary processes with extensive hemorrhages, a picture unlike that seen in acute glaucoma.

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6. It may be urged that in basing the discussion of cases of acute glaucoma with necrosis of the iris on the dialysate theory of formation of the aqueous, and calling attention to the part played by obstruction of the filtration angle in the maintenance of the high level of pressure in acute glaucoma, I am involving myself in a contradiction. This is, however, by no means the case for there is no inherent conflict between the dialysate theory and the filtration theory. I believe that as a rough approximation the dialysate theory is correct, but that superimposed on the hypothetic static equilibrium between capillary pressure and intra-ocular pressure which this theory assumes, there is under normal conditions a definite continuous circulation of the intra-ocular fluid; fluid entering the ocular cavity by filtration from the capillaries of the ciliary processes and leaving the ocular cavity by way of the canal of Schlemm. In a forthcoming paper I shall present evidence to this effect.

7. Koster: Arch. f. Augenh. **41**:30, 1895.

I repeated Koster's experiments with substantially the same results. Furthermore, careful study in serial section in my cases of acute glaucoma failed to reveal any evidence of thrombosis in the ciliary or vortex veins. I may conclude, therefore that venous stasis is not often, if ever, the cause of the capillary lesion in acute glaucoma that I have described.

2. Vasomotor Disturbance: Unfortunately, the present state of knowledge in regard to vasomotor control of the capillaries and in regard to such vasomotor disturbances as angioneurotic edema is so inadequate that there is no method by which to test this hypothesis directly. The occurrence of acute glaucoma in frequent association with emotional upsets, especially in emotionally unstable personalities, supports the notion that a vasomotor disturbance may be the precipitating cause of the attack in many cases. On the other hand, angioneurotic edema and most other evidences of a labile vasomotor system are most prominent in the pre-arteriosclerotic age, while acute glaucoma most often occurs in persons beyond middle life. I am therefore unable either to prove or to exclude vasomotor disturbance as the primary cause of acute glaucoma.[8]

3. Capillary Poison: The possible presence of some capillary poison in the eye, associated with or responsible for the capillary changes in the ciliary body, lends itself to much more direct experimental and clinical analysis. The frequent occurrence of marked chemosis of the conjunctiva and even edema of the lids in cases of acute glaucoma, while perhaps explicable as a vasomotor manifestation, is equally explicable as the result of leakage of a capillary poison out of the eye into the conjunctiva. More significant are those cases, familiar to every ophthalmic surgeon, in which operation on glaucomatous eyes has been followed by massive edema of the conjunctiva, beginning in the region of the operative wound and spreading to involve the whole conjunctiva and even the lids.

A direct experimental test for the presence of vasotoxic substances in the aqueous was possible in the following case. A woman, aged 62, was first seen when she had been suffering for four weeks from an attack of acute glaucoma. Her eye was blind, painful and congested, with a steamy cornea, a shallow anterior chamber and a dilated pupil. The intra-ocular tension was 60 mm., by the Schiötz tonometer. Under medicinal treatment no improvement occurred. One week later an

8. Sir Thomas Lewis (The Blood Vessels of the Human Skin and Their Responses, London, 1927, chaps. 14 and 15) has expressed his belief that in vasomotor disturbances associated with an altered capillary tone, histamine-like substances are released into the tissues by the nerve terminals as the result of antidromic impulses coming down the sensory nerves. The capillary reaction in this case is the direct response to the presence of the histamine-like substance in the tissues and only indirectly related to the nervous disturbance.

iridectomy was performed. For eight days following the operation the patient was free from pain, and the intra-ocular tension remained normal. On the ninth day pain, increased tension and associated signs and symptoms recurred and continued to increase in severity. Two days later the patient was at the height of an extremely severe attack of acute glaucoma, and the eyeball was enucleated under ether anesthesia.

Before the operation was started, a hypodermic needle was inserted into the anterior chamber, and 0.1 cc. of aqueous was withdrawn. This fluid and a similar amount of normal salt solution were injected at separate points intracutaneously on the flexor surface of the patient's forearm. The results of the injections are given in the accompanying table. The reaction at the site of injection of the aqueous resembled a small urticarial wheal.

A control experiment was performed on another patient who was operated on for uncomplicated senile cataract. The aqueous was col-

Diameter of Area of Swelling

Number of Minutes After Injection	Aqueous, Mm.	Salt Solution, Mm.
At time of injection	3	3.0
5	6	4.0
8	7	3.5
11	7	3.5
14	7	3.5
17	7.5	3.5
20	7	3.5
23	7	3.5
26	7	3.5
29	6	3.0

lected from the conjunctival sac immediately after the incision had been made. The site of the injection of the aqueous in this case differed in no way from that of the salt solution control. The conclusion may be drawn, then, that in some cases of acute glaucoma a vasotoxic substance is present in the aqueous, which is capable of producing an urticarial wheal elsewhere in the same patient.

If access to the filtration angle is not obstructed, the presence of a vasotoxic substance in the anterior chamber should lead to an increase in the permeability of the walls of Schlemm's canal, with a consequent increased outflow of ocular fluid and hypotension. I have therefore followed closely the course of the intra-ocular tension in cases of acute glaucoma in which relief was obtained from miotics without operation. In these cases I was frequently able to note that when the tension had begun to fall under the influence of miotics, a drop to subnormal levels of tension occurred. The period of hypotension usually lasted from six to twenty-four hours and was followed by a gradual rise to normal pressure. In some of these cases the period of hypotension was associ-

ated with an increase in the conjunctival congestion and edema (see figs. 9 and 10).

SUMMARY AND DISCUSSION

The anatomic data that have been presented indicate that acute glaucoma is regularly associated with edema of the ciliary body and with hemorrhagic, serous and fibrinous extravasations in the ciliary processes. Since no lesions of the larger vessels were found, one must conclude that the extravasations and the edema fluid are derived from the capillaries. Since capillary walls are known to be impermeable normally to the protein elements of the blood plasma, one must conclude that the permeability of the capillary walls has been increased. Such a conclusion is also necessary to explain the necrosis of the pupillary margin of the iris which is occasionally seen in acute glaucoma. The cause of this injury to the capillary walls and the peculiar localization of the lesion remains undetermined. Vasomotor disturbances cannot at present be eliminated, but in some cases of acute glaucoma a transportable vasotoxic substance is demonstrably present in the intra-ocular fluids. Whether this is the cause or the result of the glaucoma cannot at present be determined.

Two further questions of paramount importance remain to be discussed: Is the lesion in the ciliary capillaries the cause or the result of the increased intra-ocular pressure? Is the lesion in the ciliary capillaries the sole and sufficient cause of all the signs and symptoms of acute glaucoma, or are other factors needed to explain the clinical and pathologic picture of this disease? The answer to these questions may be approached in part by animal experimentation and in part by clinical and histologic studies. The experiments that I have performed in the study of these questions will be reported separately. Certain clinical and pathologic problems may, however, be briefly mentioned without any attempt at an exhaustive analysis.

1. The well known pathologic changes in the ciliary body in chronic simple glaucoma and in various types of secondary glaucoma, supplemented by my own studies in this field, indicate that the lesion in the ciliary body and ciliary processes that I have described is not present in these cases unless a severe acute exacerbation of the glaucoma has occurred shortly before enucleation. Essentially similar lesions have been seen, however, in nonglaucomatous eyes following laceration of the cornea with prolonged leakage of the aqueous. In these cases the edema and serous extravasations were not limited to the ciliary body but were found throughout the whole uveal tract and retina. Finally, I examined one patient with glaucoma that was caused by dislocation of the lens into the anterior chamber. In this case the rise of intra-ocular pressure was necessarily both abrupt in onset and extreme in degree. No edema

of the ciliary body or processes was found in this case. I may conclude, therefore, that neither the abruptness of onset of the glaucoma nor the severity and duration of the attack are responsible for the lesion, but that true acute glaucoma may occur as a complication in chronic and secondary glaucoma. On the other hand, prolonged drainage of the aqueous, with resultant extreme disparity between the intra-ocular pressure and the intra-ocular capillary pressure, may lead to serous extravasations and edema of all the intra-ocular tissues including the ciliary body. Such disparity in pressures cannot, however, be conceived of in acute glaucoma.

2. The shallow anterior chamber, the anterior subluxation of the lens and the obstruction of the filtration angle in acute glaucoma have long been attributed to the swelling of the ciliary body and processes. I am in accord with this interpretation. It should be clear that the capillary lesion in the ciliary body need not be permanent in order to explain a permanent rise in intra-ocular pressure, once the filtration angle is obstructed. If obstruction of the filtration angle is a necessary part in the development of acute glaucoma, the relative immunity of myopes with their small ciliary bodies can be understood. Likewise the curative effect of the iridectomy operation in providing that at least one portion of the filtration angle remains unobstructed is explained.

3. Both physostigmine and pilocarpine have been shown to possess the power of causing arteriolar constriction. The beneficial effect of thus decreasing capillary pressure in relation to the edema of the ciliary body is clear, altogether aside from the action of these drugs in inducing miosis and contraction of the ciliary muscle.

THE PATHOGENESIS OF ACUTE GLAUCOMA

II. EXPERIMENTAL STUDY *

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AND
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In the preceding paper evidence was presented that a lesion of the capillaries in the ciliary body and processes with resultant edema and serous, hemorrhagic and fibrinous exudations occurs constantly in acute glaucoma. The question was then raised as to which of the signs and symptoms of acute glaucoma are to be attributed directly to this lesion. The experimental procedure that was adopted to elucidate this point consisted in the injection into the eye of solutions of histamine (ergamine acid phosphate). This substance is known to be a capillary poison, causing paralysis of the capillary walls and an increased permeability of the endothelium. Solutions of varying concentration were injected in varying amounts into the anterior chamber, vitreous and ciliary body of animals of different species (dogs, cats, rabbits and monkeys). We shall present first the results of the histologic examination of these eyes, especially those in which histamine was injected into the vitreous, pointing out the similarities and differences between the lesions produced by histamine and those found in acute glaucoma. We shall then summarize the physiologic changes that were observed in these experiments.

The eyes of animals into the vitreous of which solutions of histamine had been injected were enucleated at varying intervals after the injection, fixed in Bouin solution and sectioned in paraffin. The intensity of the lesion found varied considerably from one instance to the next even when the equal amounts of histamine had been injected under as nearly identical experimental procedures as possible. In general, however, the histologic picture presented a uniformity in the character of the lesion in spite of variations in intensity. There was marked edema of the ciliary body with extravasations of serum and fibrin about the capillaries just beneath the epithelium. The ciliary processes were much swollen, especially at their tips, which were sometimes distended

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* From the Wilmer Ophthalmological Institute of the Johns Hopkins University and Hospital.

* Read in abstract at the Thirteenth International Congress of Ophthalmology, Amsterdam, 1929.

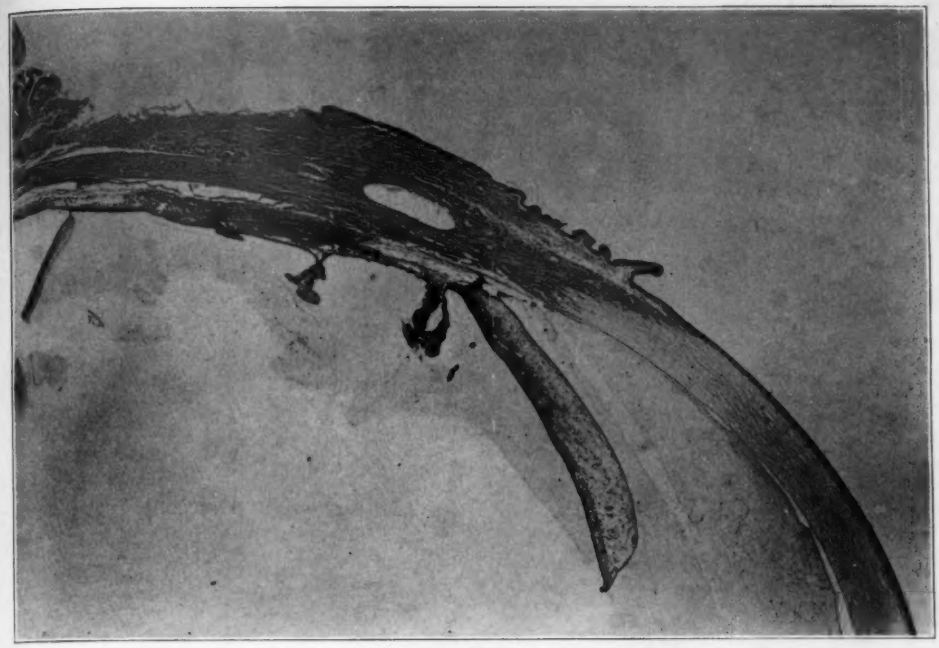

Fig. 1.—Edema of the ciliary body following the injection of histamine into the vitreous of the eye of a dog.

Fig. 2.—Edema of the ciliary processes following the injection of histamine into the vitreous.

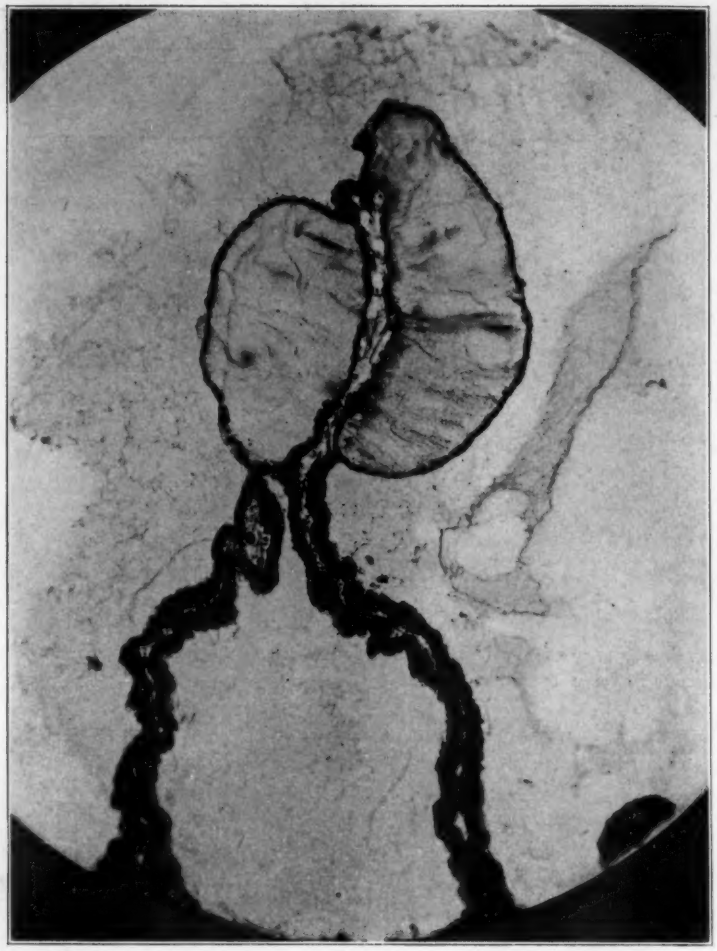

Fig. 3.—Desquamation of the epithelium of the ciliary process.

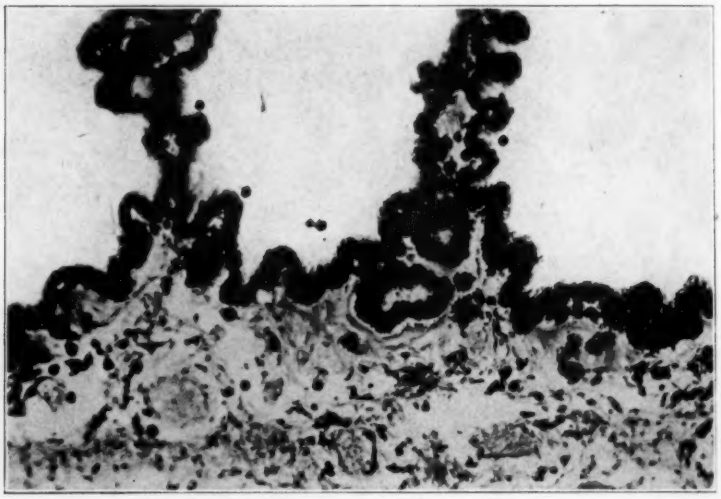

Fig. 4.—Extravasation of serum and fibrin about the vessels at the base of the ciliary processes.

so as to form great bags filled with serum and edema fluid. In places the outer layer of epithelium was raised up by a serous coagulum.

In most instances the edema of the ciliary body extended far forward, pushing the root of the iris forward. In no instance, however, was the filtration angle obstructed by the iris root. The iris showed varying degrees of edema, usually less intense than that found in the ciliary body. Coagulated serum was found in the anterior and posterior chambers. In some instances there was slight bullous keratitis, and often marked conjunctival edema. The lens showed no changes. There was often considerable serous coagulum in the anterior part of the

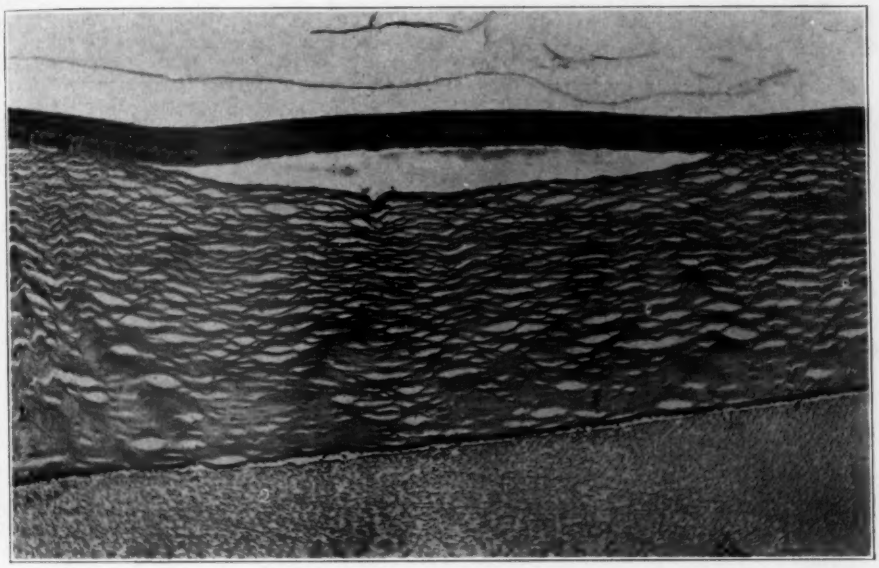

Fig. 5.—Bullous keratitis following the injection of histamine into the anterior chamber.

vitreous and occasionally some operative hemorrhage. The retina and the optic nerve were moderately edematous (figs. 1, 2, 3, 4 and 5).

In summary, the injection of histamine into the vitreous produced changes in the ciliary body and processes similar to those found in acute glaucoma. The lesion, however, was less severe and more widespread than that in human beings; it involved the iris and retina as well as the ciliary body. In no case were we able to produce obstruction of the filtration angle as the result of our injections. This, however, is not surprising in view of the relative small size of the ciliary body in the animals and the relative great depth of the anterior chamber.

The physiologic changes following the injection of histamine into the eyes varied considerably from one animal to the next. The amount of histamine injected seemed not to be responsible for these variations except in those cases in which constitutional reactions, e. g., tachycardia, low blood pressure and shallow respiration, followed from the injection of large doses (1 mg. or more). The most severe local reactions were produced by injections of 0.5 mg. or less. The degree and character of the anesthesia used also exercised an important effect, the most severe local reactions being obtained when the anesthesia was relatively light. But aside from these controllable factors, profound variations in the

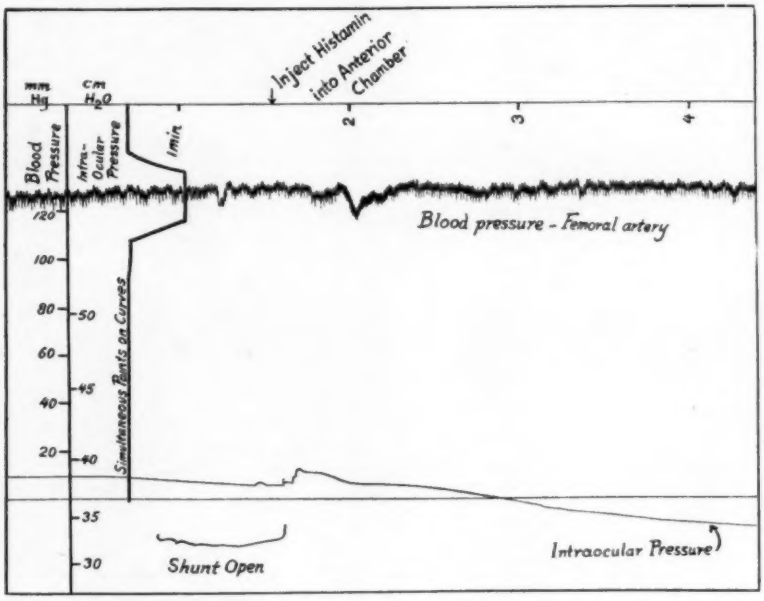

Fig. 6.—Effect of injection of histamine into the anterior chamber of a dog's eye. Paraldehyde anesthesia. Simultaneous tracings of intra-ocular and blood pressure.

degree of reaction were found in different animals of the same species that were given the same dosage of histamine under as nearly as possible identical conditions of anesthesia. The cause of these individual variations we have not been able to discover.

Injections of histamine into the anterior chamber were, in general, followed by a prompt rise of intra-ocular tension of exceedingly short duration. This was true even when, as shown in figure 6, the intra-ocular pressure was kept constant during the injection. The rise in intra-ocular pressure was followed rapidly by a drop to very low pressure, and this was accompanied by intense congestion and massive

edema of the conjunctiva, edema of the cornea and mydriasis (fig. 7). The low tension usually persisted for half an hour or more and was followed by a gradual rise to normal or slightly higher pressure.

Injections of histamine into the vitreous were, in general, followed by a prompt rise in intra-ocular pressure of much longer duration than that produced by injections into the anterior chamber. The high pressure in these cases, which lasted from ten minutes to one and one-half hours, was then followed by an abrupt fall to subnormal levels which again lasted about half an hour and was succeeded by a gradual rise to normal or slightly higher pressure. In the earlier experiments of this series the intra-ocular pressure was recorded by direct manometric measurement (fig. 8). In subsequent experiments the presence of a

Fig. 7.—Chemosis of the conjunctiva following the injection of histamine into the anterior chamber of the left eye. An equal volume of physiologic solution of sodium chloride was injected into the right eye.

needle in the anterior chamber was eliminated, and the tension was measured with a tonometer. Figure 9 represents the composite results of numerous experiments of this type, showing the cycle of changes in pressure after the injection of histamine. In the individual experiments here represented the separate phases of the reaction were present in widely varying degree. During this whole period following the injection there was usually a marked mydriasis and a decrease in the depth of the anterior chamber. The primary rise in intra-ocular pressure was entirely in harmony with what had been anticipated as the result of our knowledge of the action of histamine. The secondary fall was at first difficult to interpret. In a number of these experiments, edema of the conjunctiva and cornea was noted, and it was found that the edema always developed after the intra-ocular pressure had fallen below normal. It

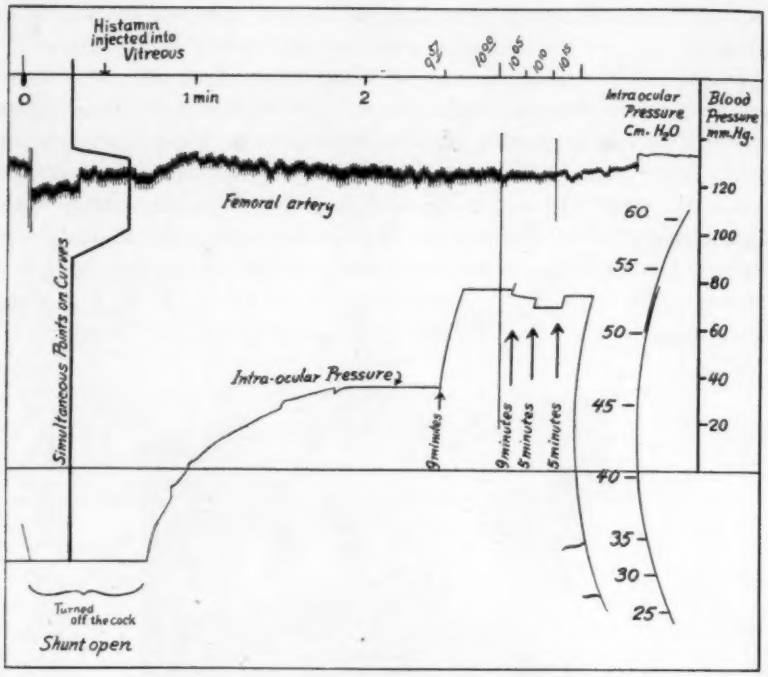

Fig. 8.—Effect of injection of histamine into vitreous of dog's eye. Paraldehyde anesthesia. Simultaneous tracings of intra-ocular and blood pressure.

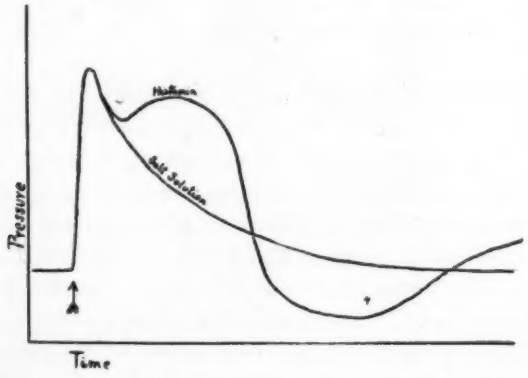

Fig. 9.—Composite curve from numerous experiments on the injection of histamine into the vitreous.

appeared, therefore, that the fall in pressure was to be attributed to the appearance of histamine in the anterior chamber and was identical with the effect previously produced by direct injections into the anterior chamber.

In order to test this hypothesis, a series of experiments was instituted in which access of the histamine to the anterior chamber was hastened or delayed by mydriasis or miosis. Under anesthesia with sodium barbital, one pupil being fully dilated by atropine and the other contracted by physostigmine, equal amounts of histamine were injected into the vitreous of each eye of dogs. The results showed that, in general, the period of high pressure was of shorter duration with atropine mydriasis than when the histamine was injected in the vitreous without previous mydriasis. On the other hand, physostigmine miosis, in general, increased the duration of the period of high pressure

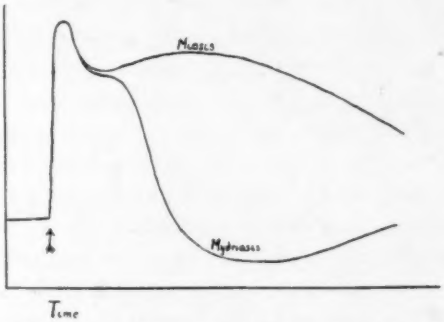

Fig. 10.—Composite curve from experiments on physostigmine miosis and atropine mydriasis followed by injection of histamine into the vitreous.

and, in some instances, abolished the succeeding period of low pressure (fig. 10). It will be remembered that the curvature of the anterior surface of the lens of dogs' eyes is much greater than that in human beings and that in marked miosis in dogs the iris presents a slightly conical form. In these experiments it was observed that the iris with a markedly contracted pupil often became slightly bombé from ten to fifteen minutes after histamine had been injected into the vitreous. The bombé condition increased until the pupillary margin of the iris was lifted away from the lens. A flow of fluid from the posterior into the anterior chamber then occurred while the iris subsided to its original conical form. Following the access of fluid from the posterior to the anterior chamber, there was often a slight drop in intra-ocular tension.

It has not been possible in these experiments to eliminate from consideration specific effects of physostigmine or atropine on the capillary reaction produced by histamine, but the observation that a slight fall in intra-ocular pressure occurred commonly after the gush of fluid through

the pupil and the subsidence of the iris bombé would strongly support the notion that the abolition of the low pressure phase in injections of histamine with miosis was due to the miosis and not to any specific action of physostigmine.

In regard to the explanation of the final rise in intra-ocular pressure, it must be borne in mind that histamine is rapidly destroyed in the tissues and that its physiologic effects are, in general, of very short duration. This late effect (one hour after the injection) is therefore not to be attributed to the histamine directly and is probably due to the presence of large amounts of serum in the aqueous.

COMMENT

These experiments are presented with the clear understanding that the experimental lesion produced differed in certain fundamental respects from that found in acute glaucoma, particularly in the short duration and spontaneous recession of the increased intra-ocular pressure. They are presented merely to indicate that certain features in the clinical and pathologic picture of acute glaucoma, in particular the shallow anterior chamber, can be produced experimentally as the result of edema of the ciliary body; while other features, in particular the chemosis of the conjunctiva, can be produced by the presence of a vasotoxic substance in the anterior chamber. The edema of the cornea in acute glaucoma is, no doubt, in large measure dependent on the increased intra-ocular pressure, but occasional cases are met with in practice in which the edema of the cornea is conspicuous while the intra-ocular pressure is only moderately elevated. These cases usually present also extreme conjunctival edema, and the picture may, therefore be, in part, the result of the presence of a vasotoxic substance in the aqueous. The same explanation may be applied to those extremely rare cases which exhibit all the usual clinical features of acute glaucoma associated with extremely low intra-ocular pressure.

The secondary fall in pressure observed in animals after the injection of histamine into the vitreous or anterior chamber also bears resemblance to the hypotensive stage which, as noted in the preceding paper, occurs not infrequently in cases of acute glaucoma relieved by miotics without operation.

STEREOSCOPIC PHOTOGRAPHY AS APPLIED TO THE EYE*

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Photography has of late years been assuming an important rôle in medicine. There are four main types applicable to the anterior structures of the eye: (1) single black and white pictures, (2) stereoscopic black and white photographs, (3) motion pictures and (4) color photography.

It is my desire to show the ease with which any one with only an elementary knowledge of photography may obtain excellent stereoscopic pictures in black and white.

The first stereoscopic photographs were black and white, taken with an ordinary camera, with great discomfort to the patient on account of the length of time and the bright light used for the focusing, and the taking of the picture. One picture was taken, then the camera was moved 6 cm. around the arc of a circle and a second picture taken. These two pictures, when viewed through a stereoscopic viewing apparatus, gave the impression of the third dimension, viz., depth. The pictures did not show sufficient detail to be of much value for small lesions of the minute structures of the eye.

The next development was a camera to take both pictures simultaneously, then the use of lenses giving magnification sufficient to show necessary detail. Attention was next directed to illumination, the problem being to have enough light for focusing the camera and taking the picture without annoying the patient. To solve this difficulty, a small pilot light was used for focusing and a shutter was placed in front of the source of illumination, as used by Dr. L. D. Redway. When the camera was focused under the dim pilot light illumination, this shutter was opened for the desired length of time (from one-tenth to one-fiftieth second). It was difficult to center the light beam accurately on the desired part. To obviate this difficulty and further simplify the apparatus, I have eliminated the pilot light and have arranged the metal-leaved shutter so that it does not entirely close, leaving a small central opening 1.5 mm. in diameter. Behind this opening a slender metal arm holds a small green filter, so that a central spot of green light is thrown on the eye for focusing. This arm also acts as a finder for the light

* Submitted for publication, Feb. 21, 1930.
* Read before the New York Academy of Medicine, Section of Ophthalmology, Jan. 20, 1930.

flash that illuminates the eye for taking the picture. When it is desired to take the picture, the camera is set for a time exposure, and the shutter on the light is snapped, the eye being exposed to the brilliant illumination for only one twenty-fifth of a second. Simultaneously with the opening of the shutter, the filter arm is kicked out of the way by a small plunger which is operated by the same pneumatic bulb that operates the shutter. The filter thus does not interfere with the illumination of the eye.

As my illuminating source I use the small Birch-Hirschfeld ultraviolet carbon arc lamp, which fits on the arm of the stand of the slit-

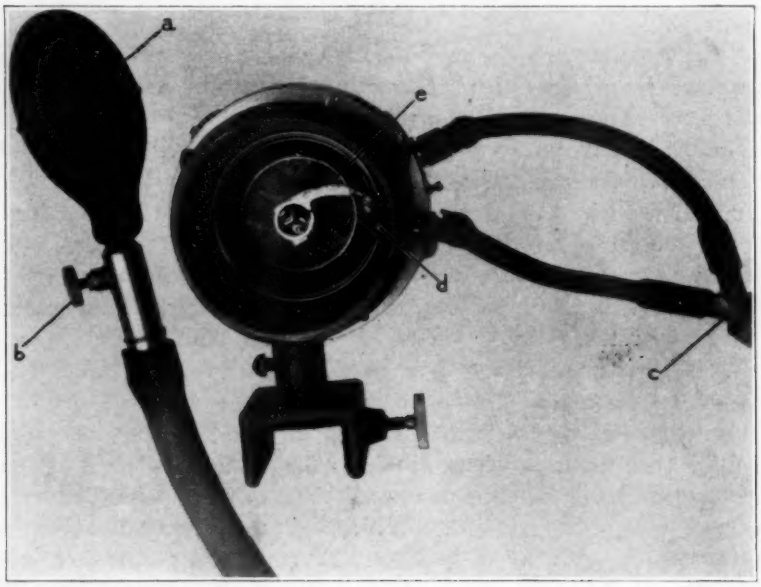

Fig. 1.—Special shutter for the carbon arc light, arranged for focusing. Pneumatic bulb (*a*) with outlet valve (*b*) connected by a Y-tube (*c*) to the plunger (*d*) operating the filter arm (*e*) and the shutter.

lamp. In the circuit of the arc, I have installed an ammeter so that the illumination may be standardized accurately at 7 amperes. The carbon arc is adjusted to emit parallel rays of light by the focusing screw. If direct current is used, the polarity must be correct; if an alternating current is used, a slightly higher amperage is necessary.

The Drüner camera replaces the corneal microscope, the low power objectives of which are fitted on the front of the camera to act as its condensing lenses. The picture resulting will be magnified one and one-half times. If higher magnification is desired, the high power

microscope objectives, replacing the low power ones, will give a picture four times natural size.

I use the Drüner rather than the anterior segment camera, because with the former a picture of the eye under oblique illumination is obtained, simulating the manner in which one illuminates the anterior part of the eye for examining it. The anterior segment camera gives a picture as though one were looking through an ophthalmoscope, which

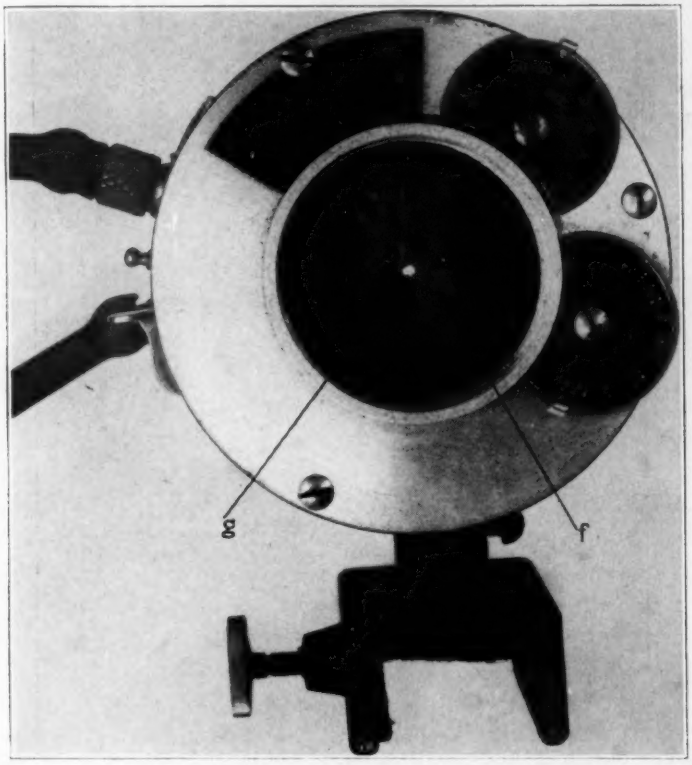

Fig. 2.—The other side of the shutter showing its metal leaves (*f*) so that it does not completely close, leaving a 1.5 mm. opening centrally (*g*).

is not the method commonly used to examine the anterior structures of the eye.

The casset which holds the two plates is placed in position on the back of the camera, the slide pulled out and the plates exposed. After exposure the casset is closed and removed to the dark room until the plates can be developed at one's convenience. When developed they are hinged together properly with isinglass. A title is made by print-

ing with india ink on a narrow strip of cellophane, which is then cemented on the emulsion side of the negative below or above one of the pictures.

The plates used are ultrarapid panchromatic, 6 cm. square. The panchromatic emulsion is preferable because, being sensitive to the whole range of the visible spectrum, all colors are correctly translated into shades of black and white, according to their light values. They

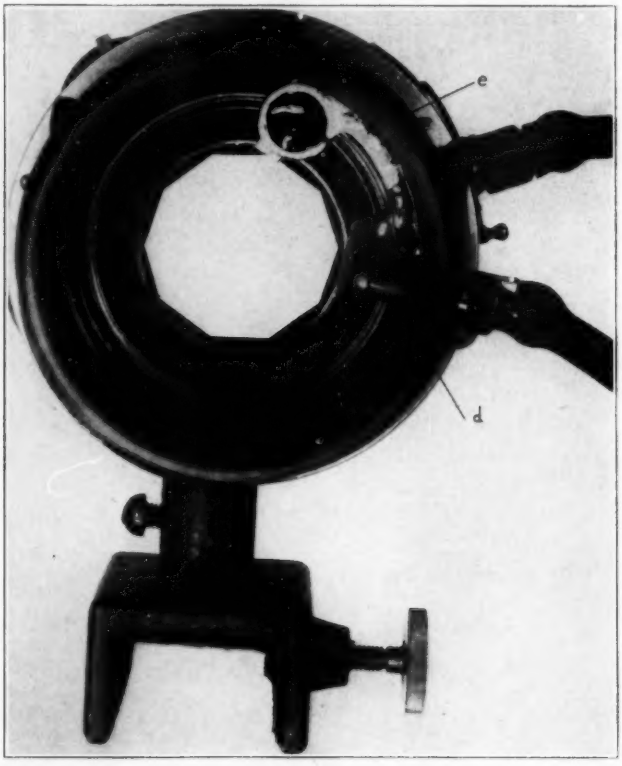

Fig. 3.—Shutter wide open with filter arm kicked to the side by the plunger. This position is held for only one-twenty-fifth second, the shutter immediately closing and the filter arm springing back to its original central position.

should be handled in absolute darkness, or if any light is needed, it should be a very dim green, as the plates are slightly less sensitive to green than to any other part of the spectrum. The ordinary orthochromatic emulsion is not sensitive to red and can therefore be handled safely in red light. It, however, does not give satisfactory results, since red photographs as black with it.

Prints are made either on paper, 6 by 13 cm., or on plates the same size to be viewed by transmitted light. The prints on photographic

paper are less expensive, more easily filed and give better results with the average negative. If the glossy no. 4 Eastman paper is used, details are brought out better than if the ordinary no. 2 paper is used. Detail is obtainable in black and white that admittedly cannot be brought out in color photography, though the color contrast, giving a pseudostereoscopic effect, considerably offsets this disadvantage.

By the use of filters of various colors in front of the carbon arc light source in taking the picture, certain colors may be diminished and others accentuated. In this manner structures of different colors, but reflecting the same amount of light photographically, may be shown up in marked contrast, which would otherwise photograph the same shade and be almost indistinguishable in the picture. I have had no experience in retouching photographic negatives or positive prints or plates.

In evaluating any procedure, its usefulness is the ultimate criterion. When considering stereophotography, one may look at it from several angles:

1. The busy oculist in his daily practice has an unbiased accurate record of lesions of the lids, conjunctiva, sclera, cornea, iris and even some conditions of the lens for future comparison. In five minutes the apparatus may be set up and a picture taken ready to be developed later.

2. The procedure is useful in teaching, and to demonstrate rare pathologic conditions the stereoscopic photograph gives a most realistic reproduction of lesions that are not always at hand for demonstration purposes when desired.

3. One is able to illustrate conditions reported at meetings, when it is impossible to have the patient himself present.

4. It can be used to illustrate articles in journals or books.

5. One can use the procedure for the study of problems in physiologic optics in regard to light transmission, light reflection, vital staining, etc., as brought out by Dr. Redway.

Details may be shown by means of stereoscopic pictures which are barely observable and meaningless in a single picture. To demonstrate this to one's satisfaction, it is necessary only to close one eye while looking at a picture through a stereoscope. Much more detail is appreciated when the two pictures are fused than when either picture is viewed separately. The advantage of a stereoscopic over a single picture is observable only by persons with a well developed fusion sense. A small percentage of observers have a fusion sense so defective that stereoscopic appreciation is impossible.

Mr. Koch, of the Carl Zeiss Company, has agreed to make duplicates of the special shutter I have devised for any one who may be interested.

LOCALIZATION OF PATHOLOGIC CHANGES IN THE CORNEA*

PROF. ERNST FUCHS

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If exogenous agents affect the cornea, it is easy to understand the localization of the changes produced, since they, as a rule, occupy the place where the obnoxious agent acted on the cornea. On the other hand, in the cases of endogenous diseases, localization often remains obscure. Why is it that in interstitial keratitis only the posterior layers of the cornea, and in keratitis nodosa the anterior ones, are affected, and in keratitis nodosa the periphery of the cornea is spared, while in other forms of dystrophies, as for instance, arcus senilis or marginal atrophy, it is exclusively involved? Therefore, one may distinguish localization according to the depth at which the changes occur (in the superficial or deep layers) from localization according to surface extension (central or peripheric)—topical localization. Different from this is the localization according to the affected histologic elements; for instance, the stroma, the corneal corpuscles or the nerves, for which I propose the term histologic localization. In many cases, for instance, in those of changes in the epithelium, in Bowman's or in Descemet's membrane, in the posterior endothelium the two forms of localization coincide. I shall demonstrate with the aid of a few instances how the differences of localization may be accounted for on the basis of the anatomy and physiology of the cornea.

EMBRYOLOGIC DEVELOPMENT OF THE CORNEA

The epithelium of the cornea, and, according to von Szily, also the lamellae, are of ectoblastic origin, while the corneal corpuscles and the endothelial cells are derived from the mesoblast. Bowman's membrane is only the modified foremost lamella, while Descemet's membrane is a cuticula developed from the endothelial cells. Therefore, these two membranes, although so alike in specimens, behave differently in pathologic cases.

Bowman's membrane is never found thicker, but often thinner or absent. Consequent to stretching, for instance in keratoconus, the membrane gradually disappears within the distended area, while ruptures, as of Descemet's membrane, are rare. Bowman's membrane possesses but

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little resistance, so that it is easily destroyed, not only by suppuration, but even by superposition of newly formed tissue like pannus or pterygium. Bowman's membrane does not regenerate.

Descemet's membrane, on the other hand, is highly resistant; it often persists even to a great extent after destruction by suppuration of the corneal stroma in its entire thickness. Scarcely ever is it found thinner, but very often thicker. If distended, it ruptures. It regenerates easily in that at the place of the defect a new cuticula develops from the endothelial cells. Descemet's membrane, being less distensible than Bowman's membrane, is also less contractile. If the cornea is reduced in size, as in an atrophic eye, Descemet's membrane is folded in a much higher degree than Bowman's. For this reason the striped opacity

Localization in the Cornea

	Topical				
Development	Chemical	Biologic	Mechanical	Adjacent Parts	Histologic
Keratoconus	Silver (argyrosis, pseudosclerosis)	A. Center free	Keratitis striata	Calcareous film	Corneal corpuscles affected; lamellae preserved
	Calcareous film	Pseudosclerosis		Pannus trachomatosa	Impregnation with metals
	Uratic acid deposits	Arcus senilis		Precipitates	Keratitis interstitialis
	Ochronosis	Lipoid infiltration		Keratitis interstitialis	Epithelium
	Dystrophia epithelialis	B. Margin free		Keratitis subsequent to scleritis	Epithelial changes in glaucoma
	Keratitis nodosa	Ulcus serpens			Nerves
	Keratitis grill-shaped	Blood staining			Herpes
		Necrosis			
		Dystrophia epithelialis			
		Keratitis nodosa			
		Keratitis interstitialis			

termed keratitis striata, which is due to the folding, is seated at the posterior, not at the anterior, surface of the cornea. During the development, the corneal tissue grows from the periphery to the center between the epithelium and the lens. Maybe because of this the fully developed cornea is thinner at the center than at the periphery. If, therefore, in the case of keratoconus the resistance of the cornea is reduced by some hitherto unknown cause, it is the central part that in the first place yields to the intra-ocular pressure.

The normal cornea is spherical. If in most eyes it appears as a horizontal ellipse, it is because above and below the limbus advances farther on the cornea than laterally. In exceptional cases the cornea is a vertical ellipse, without any interference of the limbus. In the majority of these cases, this is due to congenital syphilis influencing in this par-

ticular way the development of the cornea. Up to the present day no cases are recorded of a vertically elliptic cornea in new-born children, so that it is not known whether the normal shape of the cornea develops during fetal life or only after birth. In the latter case, the development must occur during the first year of life, as after that the cornea practically does not grow any more.

CHEMISM

The differences of the chemism of the constituents of the cornea are evidenced by their different behavior to stains, during life (vital staining) as well as after death. In the case of vital staining, the metabolism also influences the reaction to stains.

It is probable that the chemism of a determined layer is the same within its whole extent and varies only from one layer to another. For this reason changes limited to a definite layer are likely to depend on the chemical constitution of this layer. I beg to elucidate this proposition by the instance of argyrosis of the cornea.

Most frequently the silver penetrates into the cornea by direct application, whether it is consequent to long-continued instillation of a silver solution or to silver dust falling on the cornea of the silversmith. In the latter case, silver dust is also inhaled and so is absorbed; the same holds good in cases of long-continued internal use of drugs containing silver. Also ordinary food contains silver in smallest quantity, so that it gets into the circulation and may, under pathologic conditions, be deposited in certain organs, if these acquire a special chemical affinity for silver. This is the case with pseudosclerosis or Wilson's disease, in which the silver accumulates in the liver and in the cornea. In this it forms a brownish ring at the periphery of the cornea, described by Kayser and Fleischer. In this as well as in other cases of argyrosis, the silver occupies the deepest layers of the cornea and particularly Descemet's membrane, as can be seen with the slit-lamp and as has also been demonstrated anatomically by Knies. Besides Descemet's membrane, the elastic tissue has a particular affinity for silver, as is seen in specimens of an argyrotic conjunctiva in which most of the elastic fibers are stained with silver. Now, although Descemet's membrane is not an elastic tissue in the strict sense, it is closely related to it by its chemical constitution and like it accumulates silver.

In argyrosis, it is the stroma, not the corneal corpuscles, that has an affinity for silver. Other metals behave differently, being absorbed by the corpuscles and not by the lamellae, as for instance iron in cases of siderosis. With a gold stain the effect varies greatly. At the present time, staining with a solution of chloride of gold after the method of Paul Knapp is usually substituted for tattooing of the cornea. Then the gold is found to be precipitated as minute granules in the lamellae

and only much later on the corpuscles as larger granules. If rabbits are fed with sodium aurothiosulphate, so that the gold gets into the circulation, only the corpuscles stain. For the purpose of histologic examination the gold staining of the cornea is usually done by putting the cornea, immediately after death, into the gold solution. As a rule, the corpuscles alone are stained, but exceptionally it occurs that the corpuscles remain unstained, and therefore stand out light within the dark red-staining stroma. So the staining of the cornea may be either diffuse or in granules and may occur only in the corpuscles, only in the stroma or in both. This different behavior must be accounted for by variations of the chemical constitution of the constituents of the cornea. One may conclude from the foregoing that the changes produced in the cornea by metals may exhibit either a topical (deepest layers in argyrosis) or a varying histologic localization (staining of Descemet's membrane, corneal lamellae and corneal corpuscles).

The chemical constitution of the cornea changes with age and in consequence of diseases altering the metabolism.

The influence of age is evidenced by the deposit of lime in Bowman's membrane in the case of calcareous film in senile, though otherwise healthy, eyes, while in younger persons this occurs only under special pathologic conditions. The calcareous film also proves the greater predilection of Bowman's membrane for lime compared with the other layers of the cornea (topical and histologic localization). Another instance of the influence of age is the deposit of fat, forming the arcus senilis. Perhaps one may also attribute to a change of the chemical constitution by age the fact that interstitial keratitis rarely occurs in the first years of life or in advanced age.

As an instance of alteration of the chemism subsequent to general disease, I may cite the aforementioned pseudosclerosis, the deposit of pigment in the most anterior layers of the cornea in ochronosis, in which the severe changes in the joints occur, but much later, the deposit of uratic compounds in gouty persons, etc. Also anaphylaxis must be considered as an alteration of the normal chemical structure of the tissues. The anaphylaxis of the cornea is best shown by the well known experiment of Wessely. After injection of 1 or 2 drops of bovine or equine serum into the cornea of a rabbit, there follows either no reaction or a parenchymatous inflammation after an interval of fourteen days. If then, fourteen days after the first injection the same serum is injected into the other cornea, the parenchymatous inflammation begins at once. Some investigators consider interstitial keratitis resulting from syphilis as an anaphylactic inflammation; against this conception may be brought forward the fact that contrary to experimentally produced acute keratitis, interstitial keratitis is a chronic

inflammation affecting only the deep layers of the cornea, while in anaphylaxis, only the chemism of the cornea is changed in its whole thickness, and usually both eyes are affected at the same time.

METABOLISM

The metabolism of the cornea is insignificant, because the cornea has only an optical function. The metabolism is presumably most active in the epithelium, because here a permanent though slow renewal of epithelial cells takes place and because the epithelium is exposed to evaporation. Within the cornea itself the metabolism is more active in the corpuscles than in the lamellae.

Although the cornea contains much water, it is impossible to express water by pressure, because it is bound to the tissue. On the contrary, the cornea may absorb enough water to increase its thickness from two to four times. Clinically, the swelling of the cornea by imbibition from tears reveals itself as thickening and opacification frequently observed next to the border of an ulcer, particularly in children, whose cornea is more susceptible of imbibition. In the case of a perforating wound of the cornea, the imbibition is due not only to the lacrimal liquid, but also to the aqueous humor. Normally, the cornea is protected gainst imbibition with aqueous humor by its endothelial coating, as was first proved by Leber, who, after removing the endothelium of rabbits at a circumscribed place obtained opacification and swelling of the cornea, even if Descemet's membrane remained intact.

Recently, Leber's experiments were repeated by Scullia, on the eyes of dogs and man; he found that the imbibition so strong in rabbits was very slight in dogs and absent in man. Clinical observations in this respect are contradictory. Ruptures of Descemet's membrane and with it the endothelium consequent to a trauma of birth or in the course of a keratoconus are followed by swelling and opacification of the cornea. Distilled water injected into the anterior chamber causes the endothelium to be cast off, with subsequent, and sometimes permanent, opacification of the cornea. When washing out the anterior chamber for removing lens matter after extraction of cataract I always used a solution of salt isotonic with the aqueous humor and of adequate temperature; in spite of this I produced a permanent opacification of the cornea in two cases, which made me give up this procedure. On the other hand, with cyclodialysis, Descemet's membrane is often detached by the spatula to a great extent without any harmful consequences. Also incisions penetrating the cornea are, as a rule, not followed by swelling or opacification. Therefore, further observations and experiments are required for elucidation of the conditions on which the imbibition of the cornea depends after a lesion of the endothelium.

The cornea is devoid of lymph channels and therefore of a lymph current in the ordinary sense of the word. For its nutrition diffusion is sufficient, i. e., exchange of substances dissolved in the fluid at both surfaces of the cornea and in its parenchyma. Besides this, because of the evaporation at the surface of the cornea, a constant current of liquid, however slow and of however light intensity, from behind forward, must be supposed to exist. I could prove the existence of this current in a case of tattooing of the cornea after Hesse. This operation is performed by excising a disk from the superficial layers of the cornea with the trephine. The bottom of the resulting groove is covered with India ink and the removed disk is put back into its place, where, as a rule, it heals again. I examined such a case two and a half years after the operation. A uniform layer of India ink was found to lie between the posterior layers of the cornea and the reimplanted disk. From the main deposit of ink minute granules had penetrated forward and backward into the cornea, five times farther in the first direction than in the second, and as the granules of ink could have been transported only by a current of liquid, its existence was evident.

A more active metabolism exists in the periphery of the cornea because of the presence of the superficial marginal vessels in the limbus and the scant deep-seated branches of the ciliary vessels. Therefore, pathologic changes depending on the metabolism may develop differently in the central and in the peripheral parts of the cornea, as will be illustrated later.

Like the chemical structure, the metabolism also changes with age or under the influence of general diseases. The obliteration of many of the vascular loops in the limbus in old age evidences the alteration of metabolism with advancing years. Regarding general diseases, I may mention as an instance keratomalacia, which is due to avitaminosis. The amount of substances carried by diffusion to the cornea is probably the same throughout the whole extent of each individual layer. On the other hand, there is a difference in metabolism between the central and the peripheral parts because of the presence of the marginal vessels (topical localization according to surface extension). This difference may cause the pathologic changes to occur in either the marginal or the central parts of the cornea, in contradiction to the chemical constitution which determines the localization according to depth.

There are many instances of changes limited either to the periphery or to the center of the cornea. The periphery is the seat of the arcus senilis and of similar opacities accompanying proliferation at the limbus, as in cases of spring catarrh or of nevus. Then opacities are due to fat passing from the marginal vessels into the cornea where it becomes precipitated. Because of the greater number of marginal vessels in the anterior part of the cornea, the fatty deposits are numerous in the fore-

most layers. They are not contiguous with the marginal vessels, a small clear interval existing between the margin of the limbus and the arcus senilis. Beyond this clear zone the fatty opacity sets in with a sharp border and with greatest density, diminishing gradually toward the center and ending without a well defined limit. In these cases, as fat passes from the vessels into the corneal tissue, what is the factor that prevents the fat from being precipitated immediately after leaving the vessels and in their immediate neighborhood, but causes the precipitation to take place farther inward? Perhaps it is the fact that adjoining vessels, and because of them the corneal tissue, contain more oxygen and that the precipitation of the fat begins only when the amount of oxygen drops under a certain limit. The gradual reduction and final disappearance of the fatty infiltration may be accounted for by the fact that the lymph carrying the fat becomes more and more devoid of it, as it advances toward the center of the cornea. By feeding rabbits cholesterol, a ring-shaped peripheral opacity, similar to an arcus senilis, can be produced.

Also the deposit of silver in the most posterior layers of the cornea in pseudosclerosis is ring-shaped (ring of Kayser-Fleischer), and the same is true regarding the deposit of uratic compounds in the superficial layers. In all these and other similar cases the ring shape of the opacity is due to the same factors as in arcus senilis, i. e., the conditions of the metabolism, while the limitation to a determined layer depends on the chemical constitution.

There are other cases in which only the periphery of the cornea is free from changes. This should occur if, owing to the more active metabolism in the periphery, the obnoxious agents are removed instead of being brought into the cornea. This explains why a serpiginous ulcer always comes to a standstill when it approaches the limbus. The ulceration is due to the toxins of pneumococci, to combat which the normal cornea does not contain any antibodies. It is only because of the inflammation at a later date that antibodies pass from the blood into the cornea, where they enter into the peripheral parts and stop the progress of the ulcer. The leukomas resulting from a serpiginous ulcer are always separated from the limbus by a narrow transparent zone. In the cases of blood pigmentation of the cornea the minute red granules never extend as far as the limbus, because here they are dissolved and removed, owing to the more active circulation of lymph. For the same reason, the red granules are absent around the newly formed vessels within the cornea.

In necrosis of the cornea also the marginal parts are spared. A complete necrosis of the cornea is a rare occurrence (after burns or severe infections). As a rule, it is only the corpuscles that die while the

lamellae survive. The necrosis is due to the presence of toxins in the aqueous humor, developing either after necrosis of intra-ocular neoplasms or in consequence of severe intra-ocular inflammation. The toxins penetrate into the cornea from its posterior surface. In the first place, the most delicate endothelium is cast off, after which the toxins penetrate more or less deeply into the corneal stroma, in gradually diminishing concentration, where they kill only the corpuscles; whether because of a special chemical affinity for the toxins or because of their more active metabolism, the corpuscles absorb the toxins with more avidity than do the lamellae. The area within which the corpuscles become necrotic diminishes in circumference as it approaches Bowman's membrane, which, however, it reaches only exceptionally. At any rate, a marginal zone of varying breadth always remains free from necrosis. The necrosis of the cornea is a good instance of topical localization in depth as well as in surface extension and of histologic localization, as only the corpuscles are affected. When the inflammatory reaction to the necrosis has ceased, the dead corpuscles are replaced by others derived from the corpuscles in the adjoining parts. Here the corpuscles were not killed, but were irritated and brought to proliferate, and the descendants, immigrating into the necrotic area, provide it with new corpuscles. The same process takes place in the absence of toxins, if, in the case of transplantation, the transplanted piece is cut off from metabolism. Then within the first two days all the corpuscles disappear, to be very slowly replaced by the succeeding cells of the proliferating corpuscles in the adjoining parts of the cornea.

It would not do to separate strictly chemical activity from metabolism in their influence on localization. It should be assumed that the chemism of the living tissue is constantly changing because of the substances brought into the tissue by metabolism. So in certain dystrophies of the cornea (dystrophia epithelialis, nodosa [Groenouw grill-formed]) the pathologic changes are limited to the superficial layers because of their special chemism, and the marginal zone is spared because of the more active metabolism due to the marginal vessels. Some cases behave in a still more complicated way, as for instance when there is infiltration of the cornea with lipoids. This may occur in the marginal or in the central parts of the cornea. In the first case, the localization is the same as with arcus senilis and corresponds to the conditions of an otherwise normal cornea. The central infiltration is found in cases in which there previously existed changes in the center, as inflammatory infiltration or cicatricial tissue similar to the deposits of hyalin or lime, often found in old leukomas.

MECHANICAL CONDITIONS

The mechanical conditions depend on the structure of the corneal tissues and the tension to which they are exposed.

In regard to structure, the epithelium distinguishes itself from the rest of the cornea by being composed of individual cells which, in spite of their being connected by the intercellular bridges, are easily separated by liquid. Accumulation of minute droplets between the epithelial cells with subsequent opacification is a frequent condition, especially in cases of hypertonia; a condition which, in the same way, never occurs in the stroma.

The intra-ocular pressure or tension acts equally on the cornea throughout its extent, so that it may have influence on localization only if there are less resistant places in the cornea, which may become ectatic. But there is also a tension in another sense. The lamellae of the cornea are fixed in the frame formed by the margin of the sclera and are stretched within this frame by the intra-ocular pressure, so that they are permanently under a certain stretching or tension, which in a normal eye is the same in all meridians. The tension becomes unequal if an enucleated eye is compressed between the fingers in one direction. Then the lamellae are most stretched in one meridian and least in the meridian perpendicular to the first, and the result is an immediate opacity of the cornea, because the more tightly stretched parts become doubly refractive; this opacity disappears at once when the compression is relaxed. Another consequence of a difference of tension is folding. This occurs if the cornea is separated at its insertion at the sclera by a wound or an operation, as for instance extraction of cataract. Then folds form at the posterior surface of the cornea in the meridian perpendicular to the wound or incision, revealing themselves clinically as linear opacities, wrongly designated as keratitis striata. Similar radiating or parallel striation due to folds may be observed around a corneal ulcer or in atrophic eyes, if the cornea is reduced in size. The localization of such opacities is due to mechanical conditions.

NERVES

The fine ramifications of the corneal nerves have a superficial location (subepithelial and epithelial plexus). Therefore, pathologic changes dependent on the nerves must occupy the superficial layers. Their involvement in certain dystrophies of the cornea may in some measure be due to primary disease of the nerves. So dystrophia epithelialis is often preceded by an early loss of sensitiveness of the cornea, as can be verified in cases in which at first only one eye is affected with dystrophy, while in the other still apparently healthy eye the cornea has already lost its sensitiveness. In the cases of grill-like dystrophy the nerves become visible to the naked eye as fine gray lines. In certain cases of acute superficial keratitis, the minute gray efflorescences frequently appear in lines corresponding to the course of a corneal nerve.

Paralysis of the corneal nerves diminishes the faculty of the corneal epithelium to resist desiccation by retaining the water in the epithelial cells in spite of evaporation. This can be observed if the lids are kept apart with the fingers. Then it takes some time before minute grooves due to desiccation of the epithelium (Gauls) become apparent in a healthy eye. If, on the other hand, the fifth nerve is paralyzed on one side, these grooves form much sooner in the eye of this side than in the other eye.

INFLUENCE OF THE ADJACENT PARTS

In regard to pathologic changes, the area of the cornea exposed within the palpebral fissure behaves differently from the uppermost, and, in some cases, also from the lowest, part covered constantly by the lids. These parts are better protected against injuries of all kinds coming from outside and against desiccation, and they also have a higher temperature than the exposed area. According to Leber, the localization of the calcareous film within the palpebral area is due to the fact that here, owing to evaporation, the lymph in the cornea becomes more inspissated, so that the compound of lime precipitates. On the contrary, the arcus senilis usually begins above and below in the parts of the cornea covered by the lids, and the same is true regarding the artificial infiltration, similar to an arcus senilis, produced in rabbits' eyes. At present, the way by which the covering of the cornea by the lids favors the infiltration with fatty substances is not understood. On the other hand, it is easy to see why a trachomatous pannus develops at first in the uppermost part of the cornea. This is permanently covered by the upper lid, the conjunctiva of which is the potential seat of the trachomatous changes, so that by this contact at first the upper limbus is infected. It becomes swollen and broader, and finally its vessels advance into the clear cornea as pannus.

The posterior surface of the cornea is washed by the aqueous humor. With the eye open, the aqueous humor becomes cooler next to the posterior wall of the cornea by contact with the cool cornea and therefore heavier, so that it sinks to the bottom of the aqueous chamber. It is replaced by the warmer and therefore lighter, posteriorly situated aqueous humor, which is flowing upward along the anterior surface of the iris. To this permanent current of the aqueous humor together with the influence of gravity, is attributed, the localization of the precipitates in the lower part of the cornea. Also the localization of the inflammation in interstitial keratitis in the most posterior layers of the cornea may be due to the aqueous humor, if one assumes that from it the obnoxious agents act on the cornea—topical localization. In these cases there is also a histologic localization in that the exudate composed of lymphocytes

is the product of the proliferation of the corneal corpuscles, which therefore possess a greater affinity for the syphilitic virus than the lamellae. Also in cases of deep keratitis the localization in certain parts of the cornea is often determined by the place at which an exudate in the anterior chamber is in contact with the cornea.

The cornea borders on the sclera, uvea and conjunctiva, so that diseases of these tissues encroach at first on the peripheral parts of the cornea.

I have attempted to make clear in a few instances the factors that must be taken into consideration in determining the cause of the localization of pathologic changes in the cornea.

Ophthalmologic Review

KRUKENBERG'S SPINDLE*

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PHILADELPHIA

Krukenberg, in 1899, was the first to report this unusual type of pigmentation of the cornea. He described the lesion as brown, spindle-shaped and symmetrical and occupying the deepest layers of the cornea. The line of pigment ran in a vertical direction, and he referred to it as "bilateral congenital melanosis of the cornea."

He described three cases, in all of which myopia was found, ranging from —1 to —9 diopters. The patients were all women over 45 years of age. Each had brown irides. Krukenberg expressed his belief that this condition occurred only in brown eyes. The size of the pigmented spindles varied from 4 by 3 mm. to 3 by 4.5 mm. No adhesions or precipitates were seen, and the pigmentation of the cornea and irides was of the same color. Two of his patients had floating vitreous opacities and one had a bilateral posterior staphyloma.

He concluded that in fetal life when the pupillary membrane was contiguous with the cornea the pigment was fused with the cornea, and that the similarity in color between the pigmented area and the iris indicated some connection. He believed that intra-uterine inflammation could be excluded as a causative factor because of the bilateral deposits in each cornea, their location and symmetry and the absence of evidence of a pupillary membrane. There was no history of any inflammation in any of these cases, nor was there any remnant of persistent pupillary membrane.

In 1901, Stock reported a case of pigmentation of the cornea in a man, aged 61, with myopia of —15 diopters. In this case there were also incipient cataracts, vitreous opacities and myopic changes in the fundus. The right disk was pale and the nasal field contracted.

The pigment of the cornea was described as reddish brown, finely granular and forming a vertical spindle in the deeper corneal layers. The spindle pigmentation measured 3 by 4 mm. The color of the irides was brown, and the anterior chambers were normal. The spindle in the left eye was external to the center of the cornea and was central

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in the right eye. There was no history of any inflammatory condition or evidence of persistent pupillary membrane.

Stock regarded the pigmentation of the cornea as an anomaly in that the cells remaining on the posterior surface of the cornea after formation of the anterior chamber had the power of developing pigment at the time of birth. He added the history of another case, that of a woman, aged 40, with high myopia and gray irides, who had a gray spindle-shaped opacity in the deeper layers of the cornea, which was seen to be made up of fine dots. He suggested as a name for this type "corneale membrana pupillaris preservans," leaving the term "congenital melanosis of the cornea" for the highly pigmented type, as suggested by Krukenberg.

In 1903, Thompson and Ballantyne reported a similar case of pigmentation of the cornea in a woman, aged 22. The patient was a myope with corrected vision of 6/9 in each eye. In each cornea was a faint brown, vertical streak of pigment. Below the center of the cornea, the pigment spread out like the tail of a comet. This was thought to be in the stroma, but its exact depth could not be determined. The size of the spindle was not mentioned.

In this case, examination revealed the irides to be light with a partial coloboma of each disk. There was no evidence or history of a postinflammatory condition, nor was there any trace of a persistent pupillary membrane.

The authors regarded the etiology as indefinite, but they believed the cause to be a constant one.

Kraemer, in 1906, cited the history of a nervous patient, a woman, aged 63, who complained of failing vision which was due to a bilateral postneuritic atrophy. In the right eye, vision was 5/18; with a +1.25 cylinder axis 10, it was 5/5. In the left eye, vision was 5/12; with a +1.10 cylinder axis 10, it was 5/5.

This case showed hyperopic astigmatism with a concentric contraction of the fields for form and color. There was no history of inflammatory disturbance, nor was there any trace of pupillary membrane or Schubert's lens dust.

Of special interest is the fact that each cornea revealed a fine brown pigmentation, in the form of a spindle, lying directly in front of the middle of the pupil but horizontally situated in the posterior layers of the cornea. This is the only case reported in which the spindle was in the horizontal direction. The border of the spindle was more densely pigmented than the center, and a gray haze was noted around the pigment granules. The size of the spindle was not mentioned. The irides were steel gray. The anterior chambers were normal and the pupillary borders sharp.

Kraemer accepted Krukenberg's explanation of this pigment, and he further suggested that a union could occur between the membranes in any direction, and that this would explain the horizontal position of the pigmented spindle.

Holloway, in 1910, reported three cases of Krukenberg's spindle, the first to be recorded in this country.

His first patient was a woman, aged 32, who complained of a constant dull aching pain in each eye. The family history was irrelevant. Her health had been good, except for an attack of intermittent fever many years before. She had worn glasses for eight years. There was no history of trauma or ocular inflammation. The pupils were equal and round and reacted normally. Ocular rotations were full.

By oblique examination and the loupe, a spindle-shaped, vertical streak of pigment could be seen, which was golden brown and situated in the deeper layers of the cornea; their exact depth could not be determined. The central dots were more compact than at the periphery, and the adjacent cornea was clear. It measured 2.5 by 1 mm. There was no evidence of pupillary membrane. The irides were dark brown. The anterior chambers were normal, and the media and fundi were clear.

With the ophthalmoscope, a faint central, vertical linear opacity could be seen, with a faintly granular condition of the streak. Both eyes were the same, except that the streak in the left eye was just external to the center of the cornea.

Vision was 6/6 in each eye. In the right eye, refraction with a +0.25 sphere +0.50 cylinder axis 30, showed a vision of 6/4; in the left eye, with a —0.25 sphere +1.25 cylinder axis 180, vision was 6/5.

The second case was that of a woman, aged 32, who came to the clinic complaining of symptoms of eye strain. The family history and the personal history were negative. She stated that she had had styes in childhood but that otherwise her eyes had given her no trouble. She had worn glasses for ten years. The pupils were equal, round and reacted normally. Ocular rotations were normal.

With oblique illumination and the loupe, the streak was seen to be golden brown, as in the first case, and the granular condition could be best seen at the periphery, running in a vertical direction. The spindle measured 3 by 1 mm., and was located deep in the stroma. The other portions of the cornea were clear, and there was no evidence of persistent pupillary membrane. The anterior chambers were of normal depth. The irides were bluish gray and the media and fundi were clear. Vision was 6/9 in each eye. In the right eye refraction with a —1.00 sphere —0.50 cylinder axis 120 gave a vision of 6/6; in the left eye, with a —1.25 sphere —0.25 cylinder axis 60, vision was 6/6.

The third case was that of a woman, aged 40, who had always been myopic and who had worn glasses for twenty-five years. The personal

and family histories were irrelevant. There was no history of any ocular inflammation or trauma. In the stroma of each cornea, just at the inner side, was a brownish, granular area, more dense in the center and running in a vertical direction. The spindle measured 3 by 1 mm., and the adjacent cornea was clear. The pupillary reactions were prompt and full, and the media and fundi were normal. The iris was bluish gray. Vision was 6/45 in each eye. In the right eye, refraction with a —3.50 sphere —1.00, cylinder axis 165, showed vision to be 6/6; in the left eye, with a —5.00 sphere —1.50, cylinder axis 15, vision was 6/5.

In the cases reported by Holloway there was some variation in the size of the pigment dots. He did not present any theory regarding the origin of the spindle.

Tweedie, in 1911, reported a case of congenital pigmentation of the cornea in a man, aged 25. The personal history was irrelevant. There was no history of any eye trouble except a nebula on the right cornea which had been present since birth.

Oblique examination revealed a central spindle, slightly curved, on the right cornea. The left eye showed the same condition, except that the pigment was not quite so dense and was located to the nasal side of the center. The pigmentation was yellowish brown. The dots were more numerous in the center and became smaller and more scattered toward the periphery. The size spindle was 2.5 by 3 mm., and was located deep beneath Bowman's membrane. The rest of the cornea was clear. The anterior chambers were normal. There were no remnants of pupillary membrane. The vitreous and the fundi were clear. There was a slight nystagmus when the eyes were turned to the left. The patient had moderate myopia. The right eye was tested for refraction with a —3.75, cylinder axis 180; the left eye with a —1.75 sphere +2.75, cylinder axis 97.5.

Augstein, in 1912, reported six cases seen in 12,000 patients examined, but he did not report their cases in detail. In his six cases the spindle pigmentations were exactly the type described by Krukenberg and Stock. Each patient had a vertical, brown spindle pigmentation lying on Descemet's membrane, which did not enter the corneal lamellae. None of them showed any formation of threads on the iris. He stated that this pigment spindle had nothing to do with the membrana pupillaris perservans.

Mills, in 1913, reported a case which showed a vertical, spindle-shaped, brownish deposit in the deeper layers of both corneas, consisting of minute dots, which were dense at the center and more isolated at the margins. There was no evidence of any previous inflammation. The symmetry and the characteristic appearance justified the conclusion that the condition was a congenital melanosis of the cornea. Mills agreed with Krukenberg and Stock that at a certain period of develop-

ment the pupillary membrane is in contact with the cornea and becomes absorbed but the pigment remains and penetrates the cornea. This theory is borne out by cases with a persistent pupillary membrane connecting the cornea with the iris.

Kraupa, in 1917, reported three cases in women, all of whom had a high degree of myopia. The ages varied from 56 to 60. Each patient showed fine pigment dust in the deepest layers of the cornea, forming a spindle of the type described by Krukenberg. Two of the cases showed fine pigment dust scattered over the iris. All the patients had brown irides, and in one case the spindle was unilateral.

Kraupa stated that some cases of melanosis that have been described as congenital are possibly pathologic, and he believed that congenital and acquired endothelial pigmentations were similar. He agreed with Krukenberg and Seefelder that the origin of the pigment was in the pupillary membrane. He stated that he had found it in the deepest layer of the cornea, as well as in the anterior capsule of the lens and iris at the same time.

One of his patients presented herself for relief from acute glaucoma. The other patients complained of poor vision.

Vogt, in 1921, reported four cases, in all of which the patient was over 35 years of age. He found that the pigmentation was often associated with endothelial elevations. In one case there was pigment dust over the entire posterior surface of the cornea, and in another the spindle merged with finer pigment granules on each side. Vogt expressed the belief that there are all stages between diffuse pigmentation and the formation of the spindle. He stated that a marked depigmentation of the iris existed in all the cases that he studied, and he classified it as a senile change of the endothelium in the axial area, causing the granules freed in this depigmentation process to adhere to the endothelium of the cornea. According to Koby, Vogt also stated that it is usually bilateral and that there is complete independence from the persistent pupillary membrane. Further, it is constantly associated with myopia and is usually fusiform. There is axial distribution of pigment, which reaches neither the inferior nor the superior limbus, and the pigment appears only in old people. He believed the spindle pigmentation to be acquired.

Ezell, in 1922, reported a case in a book-keeper aged 60. The personal and family histories were irrelevant. In both eyes a pigment spindle ran vertically through the center of the cornea. The pigment was most dense opposite the pupil, but it traversed the whole diameter of the cornea. On each side of the spindle, the cornea was clear. The pupils were normal, and they reacted equally to light and in accommodation. Ophthalmoscopic examination gave normal results except for myopia. The visual fields were slightly contracted above and below.

Color sense was normal. The right eye was tested for refraction with a —14.00 sphere, and the left eye with a —15.00 sphere.

Ezell concluded that melanosis of the cornea may be either congenital or acquired. The form and the location of the pigmented area are quite different in the two types. If congenital, the pigment appears in the form of a vertical oval, affecting the deeper layers of the cornea in its central portion which looks as if it had been painted with brown or black paint. The condition is usually symmetrical in both eyes and is more common in women than in men. In the acquired type, the pigment does not assume any special form. The cornea may be pigmented in a small area or be entirely covered. The posterior endothelial layer may be pigmented or the pigment may penetrate the substance of the cornea. Ezell expressed the belief that congenital melanosis is due to an abnormal development of the uveal tract. Some authors attribute it to multiple sclerosis, since it has been observed in several such cases. Salus attributed it to this cause. According to Yamaguchi, primary melanosis of the cornea is rare. In acquired melanosis a collapsed anterior chamber, a wrinkling or folding of Descemet's membrane and a loss of endothelium are predisposing factors. The condition may develop when the anterior chamber has been empty a long while and the iris remains in contact with the cornea. The iridic pigment is then deposited on the posterior surface of the cornea.

Hanssen, in 1923, described the case of a woman who was brought to the hospital for gas poisoning and who died there. A careful examination of the eyes revealed a central corneal pigmentation on both sides, in the typical form of Krukenberg's pigment spindle. The patient was not aware of its existence. There were no signs of actual or previous inflammation. The media and the fundi were normal.

Microscopic examination showed that at the site of the spindle the posterior wall of the cornea was pigmented and that the endothelial cells, which elsewhere were regularly formed and situated, were at this point thickly filled with pigment dust. Therefore, as von Hippel found, the endothelial cells must here have developed phagocytic properties. Moreover, the framework of the angle of the chamber was found infiltrated with fine free pigment. The lumina of Schlemm's canal were surrounded in part by particles of pigment. At the end of Descemet's membrane there was also pigment in some places in front of the membrane, which had evidently migrated from the angle of the chamber. It could not be determined whether this was a congenital anomaly. Hanssen thought that the origin of the pigment was in the iris.

In 1926, Cardell reported the case of a woman, aged 30, in whom there was no family history of myopia or of any previous ocular inflammation. The cornea presented a vertical, spindle-shaped pigmentation symmetrically arranged in each eye, situated below the horizontal

meridian and 1.5 by 3 mm. in size. The pupils reacted normally to light. The irides were gray and showed no sign of persistent pupillary membrane. The left eye was more myopic than the right, and showed an area of choroidal atrophy at the outer side of the disk. The right eye was tested for refraction with a —10.00 sphere, and the left eye with a —20.00 sphere.

Slit-lamp study revealed the pigment on the posterior layers of the cornea. It was composed of multitudes of closely set brown rings.

This case was reported at a meeting of the British Ophthalmological Society, and there was some discussion as to whether the pigment was congenital or not. Thompson and Ballantyne, in support of the former view, said that the pigmented areas were of perfect symmetry, the spots of the same size and the eyes myopic. They further emphasized the absence of a previous inflammation, and stated that the pigmentation was interstitial and not on either surface of the cornea.

Microscopic section in this case "shows the pigment to be in Descemet's membrane, immediately under the cubical endothelium, beginning at a little distance from the angle of the anterior chamber, where the limitation of the pigmentation is sharply defined and thence fades toward the center of the cornea."

In 1926, Gifford reported a case of endothelial dystrophy of the cornea, which also had a spindle-shaped pigmentation and exactly resembled the so-called Krukenberg spindle. In this case, which was in a woman, aged 31, the pigmentation was bilateral and ran in a vertical direction. Slit-lamp study revealed that the pigment spindle was located on the endothelial surface, and that it resembled deposits of bronze. The pigment was thicker in the center of the spindle, less dense in the periphery and associated with endothelial elevations. There were no signs of present or past ocular inflammation. Gifford did not give the refractive error. The unusual feature was the fact that it was associated with endothelial elevations. Vogt discussed the relation of this condition, endothelial elevations, to Krukenberg's spindle, and explained the elevations as being closely set Henle's warts of Descemet's membrane.

Peter, in 1927, presented a case of bilateral pigmentation on the posterior surface of each cornea, in a woman, aged 54, a hyperope. The pigmented spindle occupied the center of the pupillary area and ran in a vertical direction. With a plus 20 sphere in the ophthalmoscope, the spindle was seen as a dark brown linear precipitate. The slit-lamp revealed a fusiform pigmented line. The precipitates were red and stellate, and in all probability were washed out and scattered over the surface by the aqueous, because the lines did not extend to the limbus from above or below. Peter stated that Krukenberg and Vogt described this rare type of corneal pigmentation known as Krukenberg's spindle.

Koby, in 1927, reported two cases in which the condition developed in patients who were under his care. The first was in a woman who had iritis. Four months after her recovery, a pigmented spindle was seen to be forming in one eye; there was no spindle in the other eye. In the second case, which occurred in a man, the pigment dots were seen on the posterior surface of the right cornea, which in one and one-half years developed into a typical spindle of the Krukenberg type. There were no signs of ocular inflammation in this case.

Koby believed that the condition was due to convection currents in the anterior chamber, and he agreed with the theory advanced by Turk that the granules scratched the delicate endothelium and so facilitated deposition. He stated that the reason the center of the spindle was below the center of the cornea was the action of gravity. Disintegration of pigment is facilitated by myopia, senility and chronic inflammation, and the spindle is quicker to form, if due to an inflammatory process.

If this conclusion is accepted and if his cases were typical cases of the Krukenberg type, it is apparent that this peculiar corneal pigmentation may be acquired.

The first case reported by Koby was somewhat similar to that reported by Mules, in which the center of the pigmented area was below the center of the pupil, but he did not consider it a type referred to by Krukenberg. Mules' case was one of double symmetrical opacity of each cornea, in a man, aged 70. The personal history was irrelevant. The pigment was chocolate brown and ran in the vertical meridian of each cornea. It was in the form of a rounded cone, the apex being at the center of the pupil and the base lying on the edge of the cornea, occupying about one fifth of the diameter of the cornea. It caused the patient no inconvenience, and he was not aware of its existence. Mules stated that he had watched this condition develop for a period of eight years. This is a case of acquired melanosis of the cornea but it is not of the Krukenberg type.

James, in 1927, reported a case of Krukenberg's spindle in a woman, aged 63, who came for the removal of a cataract. She had a high degree of myopia. The right eye was tested for refraction with a —14.00 sphere, and the left with a —10.00 sphere.

The spindle was present in the right eye only. The left eye was cataractous and divergent and showed retinal detachment with choroidal atrophy. In the right eye there was an oval pigmentation, running vertically, slightly toward the nasal side. It consisted of fine brown dots and, with the loupe, looked as if it was deep in the cornea. The appearance of the pigment was like the peppery deposits seen on the anterior surface of the lens in cases of persistent pupillary membrane. The pupil was evenly dilated, and there were no tags present. With the ophthalmoscope, a large myopic crescent was noted. This was the only pathologic lesion seen in this eye. With the slit-lamp the change in the

cornea appeared to be more diffuse than one would be led to expect by examination with the loupe and the condensing lens. With the latter, the appearance was really that of a spindle, more or less in the center of the cornea. According to James' drawing of the pigmentation, the spindle seemed to run horizontally as well as vertically and formed a cross, but was more diffuse in the vertical direction.

Goar, in 1928, reported the case of a myopic patient, a man, aged 40, with bluish-gray irides. In the right eye, refraction with a —0.75 sphere —1.00, cylinder axis 50, and in the left eye, with a —1.50 sphere, showed vision to be 6/6.

Careful oblique examination revealed a fusiform pigmentation in the deepest layers of the cornea. The long axis of the spindles was in a vertical direction but slightly nasal to the center in each eye. Slit-lamp study showed the spindles to consist of closely packed dots of chocolate-brown granules lying on Descemet's membrane or on the endothelial cells of the cornea. Viewed in the axis of specular reflection, the pigment dots seemed to lie in and between the endothelial cells. The pigmentation was considerably more diffuse than one would suspect from the examination with the naked eye or with the loupe.

Outside of the spindle, apparently, the cornea was not clear. The pigmentation extended toward the periphery of the cornea above but was much less dense than at the center of the spindle, while below the dots reached the limbus, flaring out laterally and mesially, partially to encircle the cornea close to the limbus. The other eye was similar. There was no evidence of pupillary membrane or other anomaly.

In January, 1929, Friedman reported a case in a man, aged 32, with fusiform deposits lying in or on the posterior corneal layers, not quite in a vertical direction, as the upper end of the spindle leaned toward the temple about 15 degrees. The spindle was similar in the other eye. Its length was about two thirds of the corneal diameter, and the center of the spindle was about 1.5 mm. below the corneal center. Slit-lamp study revealed a mass of chocolate-brown pigment dots, larger in the center and smaller at the periphery. The dots were circular and flat, and lay behind Descemet's membrane on the endothelial layer. The irides were bluish gray and showed no signs of ocular inflammation. Over the surface of the iris, clumps of brown pigment were scattered, the largest masses being in the crevices of the muscle bundles. The right eye showed remnants of a persistent pupillary membrane. There was none in the left eye, and there was no other abnormality. The patient had myopia of slight degree. He showed eccentric fixation, although the fields were normal. Probably in this case the central spindles interfered with vision.

Friedman was inclined to agree with Koby that the spindle is not due to a congenital anomaly or developmental defect but is the result of some inflammatory process.

In the literature I have noted no other case of Krukenberg's spindle associated with a pupillary membrane or in which vision was impaired.

Korobova, in 1929, reported a case in a man, aged 50. The pigmentation, in a vertical direction, was noted in the right eye. The left eye was clear. Several months later, a beginning pigmentation was observed in the left cornea, also running in a vertical direction. The left eye was subsequently removed because of painful uveitis and complicated cataract. Microscopic section showed proliferative and degenerative alteration in the pigment epithelium of the eye. There was liberation of pigment granules, and their migration into the corneal endothelium was noted.

Korobova believed that the origin of Krukenberg's spindle is an inflammatory process and that pigment granules are set free from the intra-ocular epithelium. They are then transferred by the lymph current into the anterior chamber, assuming a vertical line. Finally, through the phagocytic action of the corneal endothelium, they are fixed as a vertical spindle on the posterior surface of the cornea.

In November, 1929, Griscom and Tassman reported the case of a colored woman, aged 36, with a vertical pigmentation on the posterior surface of each cornea. She presented herself for relief from pain in her eyes. Vision was 4/40 in the right eye and 1/40 in the left. With correction her vision was 5/40 in each eye. The patient was a myope. Her family history was irrelevant, and there was no history of any ocular inflammation. External examination revealed unequal pupils which did not react to light. The anterior chambers were deep. The tension was normal and convergence was poor. Oblique examination showed a linear, brown streak in the center of each cornea, extending vertically from just below the upper limbus to within 1 mm. of the lower limbus. With the ophthalmoscope there could be seen in each eye a brown, fusiform streak consisting of a number of small, closely placed, individual dots. No remnant of pupillary membrane was seen. The lens showed star-shaped striations and numerous floating vitreous opacities. The disk was pale. The macular region revealed a large area of degeneration, surrounded by pigment heaping, and there were several areas of choroidal atrophy throughout the fundus.

The Wassermann and Meinicke reactions were strongly positive. The systolic blood pressure was 110 and the diastolic 60. Roentgenograms of the sinuses showed the ethmoids and the right maxillary sinus to be clouded. Roentgenograms of the teeth revealed moderate absorption of the alveolar processes and several root stumps with infection about them.

Slit-lamp examination showed a palisade arrangement of dark brown pigment all around the limbus. The anterior portions of the corneas were clear. Scattered over the entire posterior surfaces of each cornea were fine, brown granules; these were more densely arranged

Fig. 1.—Krukenberg's spindle, as seen with the loupe and the condensing lens.

Fig. 2.—Krukenberg's spindle, viewed with slit-lamp.

Fig. 3.—Corneal prism of spindle (slit-lamp).

in the center of each cornea to form a vertically oval configuration. This pigment extended from just above the lower limbus to about halfway between the center and the limbus above. Most of the pigment dots were in front of the endothelial cells, but it was thought that some of them might be behind this layer. This iris was markedly atrophied in each eye. The anterior surface of each lens contained some fine, brown pigment granules, which resembled those deposited on the cornea.

I shall report what I believe, after an exhaustive search in the literature on this subject, to be the thirty-sixth case.

REPORT OF CASE

V. P., aged 38, an Italian laborer, appeared at Dr. Shannon's clinic, at Jefferson Hospital, in June, 1929, for relief from a pterygium on the left eye. The personal and family histories were irrelevant. There was no history of injury or ocular inflammation, and there was no disturbance of central vision.

In both eyes vision was 6/6, and refraction with a +50 sphere, cylinder axis 180 gave a vision of 6/5.

The lids, lashes, conjunctiva and tear sacs were normal. Ocular rotations were full in all directions, and the eyes were out slightly under cover. The tension was normal, and the anterior chambers were of the usual depth. The pupils were 2 mm. in diameter, equal and round, and they reacted normally to light and in accommodation. The irides were brown. The ophthalmoscopic examination revealed no synechiae or vitreous opacities. The disks in each eye were of good color, and the edges were sharp and well defined. The macular areas were normal. The vessels and the results of a general fundus examination were normal. The form and color fields were within normal limits. There were no central or peripheral scotomas, and the color sense was normal. The Wassermann and Kahn reactions were negative. The urine was normal, as were the teeth and sinuses from both a clinical and a roentgen standpoint. The results of a general physical examination were negative, except that the tonsils were found to be diseased. No other condition was found pointing to any pathologic process. The blood pressure was 130 systolic.

Routine examination revealed a vertical pigmented deposit in the center of each cornea. By oblique examination a faint band of brown pigment could be seen which appeared at first to be in the substantia propria of the cornea. The ophthalmoscope, however, showed this pigmentation to be deeper. Slit-lamp study further revealed these brown granules of pigment to be lying in front of the endothelial layer and intimately attached to it. The dots were golden brown and varied slightly in size, their arrangement forming a spindle-shaped mass. The spindle streak, 3.5 by 1 mm., in each cornea was directly in front of the pupil and leaned slightly toward the temple, with the greatest diameter of the pigmented area below the horizontal midline. In the thicker parts of the spindle it was difficult to determine whether the pigment was posterior to the endothelial layer or in front of it, but at the edges of the mass it was easier to determine their location. The slit-lamp showed this pigmentation to be spread out over a greater area than was observed by oblique illumination or by the ophthalmoscope. Below the spindle the pigment dots spread out like a fan and could be seen extending almost to the lower periphery of the cornea. The pigment extended above, just beyond the lower border of the iris. The dots were compact in the center of

	Date	Bilateral	Location	History of Inflammation	Color of Pigment	Symmetrical	Shape of Spindle	Pupillary Membrane	Vertical Spindle	Vision After Correction	Congenital Defects
1. Krukenberg	1899	Yes	Central	No	Brown	Yes	Oval	No	Yes	.....	None
2. Krukenberg	1899	Yes	Central	No	Brown	Yes	Oval	No	Yes	.....	None
3. Krukenberg	1899	Yes	Central	No	Brown	Yes	Oval	No	Yes	.....	Cupping of disk
4. Stock	1901	Yes	External to center	No	Reddish-brown	Yes	Oval	No	Yes	Fingers at 4 M.	None
5. Thompson and Ballantyne	1903	Yes	Central	No	Brown	Yes	Oval	No	Horizontal	6/9	Partial colobeoma of disk
6. Kraemer	1906	Yes	Central	No	Brown	Yes	Oval; lower part spread out	No	Yes	5/5, 5/5	.....
7. Holloway	1910	Yes	External to center in right eye	No	Golden brown	Yes	Oval	No	Yes	6/5, 6/4	.....
8. Holloway	1910	Yes	Central	No	Golden brown	Yes	Oval	No	Yes	6/6, 6/6	.....
9. Holloway	1910	Yes	Internal to center	No	Brown	Yes	Oval	No	Yes	6/6, 6/5	.....
10. Tweedie	1911	Yes	Internal to center	No	Yellowish-brown	Yes	Oval	No	Yes	6/6, 6/6	Nebula on the right side of cornea
11. Augstein	1912	Yes	Central	No	Brown	Yes	Oval	No	Yes	.....	.....
12. Augstein	1912	Yes	Central	No	Brown	Yes	Oval	No	Yes	.....	.....
13. Augstein	1912	Yes	Central	No	Brown	Yes	Oval	No	Yes	.....	.....
14. Augstein	1912	Yes	Central	No	Brown	Yes	Oval	No	Yes	.....	.....
15. Augstein	1912	Yes	Central	No	Brown	Yes	Oval	No	Yes	.....	.....
16. Augstein	1912	Yes	Central	No	Brown	Yes	Oval	No	Yes	.....	.....
17. Mills	1917	Yes	Central	No	Brown	Yes	Oval	No	Yes	.....	.....
18. Kraupa	1917	Yes	Central	No	Brown	Yes	Oval	No	Yes	.....	Nebula on left side of cornea
19. Kraupa	1917	Yes	Central	No	Brown	Yes	Oval	.....	.....	.....	.....
20. Kraupa	1917	No	Central	No	Brown	No	Oval	.....	.....	.....	.....
21. Vogt	1921	Yes	Central	.....	Brown	.....	Oval	.....	.....	.....	.....
22. Vogt	1921	Yes	Central	.....	Brown	.....	Oval	.....	.....	.....	.....
23. Vogt	1921	Yes	Central	.....	Brown	.....	Oval	.....	.....	.....	.....
24. Vogt	1921	Yes	Central	.....	Brown	.....	Oval	.....	.....	.....	.....
25. Ezell	1922	Yes	Central	No	Brown	Yes	Oval	.....	.....	.....	.....
26. Hanssen	1923	Yes	Central	No	Brown	Yes	Oval	.....	.....	.....	.....
27. Cardell	1926	Yes	Central	No	Brown	Yes	Oval	.....	.....	.....	.....
28. Gifford	1926	Yes	Central	No	Brown	Yes	Oval	.....	.....	.....	.....
29. Peter	1927	Yes	Central	No	Dark brown	Yes	Oval	No	Yes	.....	.....
30. Koby	1927	No	Central	Iritis	Brown	No	Oval	No	Yes	.....	.....
31. Koby	1927	No	Central	No	Brown	No	Oval	No	Yes	.....	.....
32. James	1927	No	Nasal side of center	No	Brown	No	Oval	No	Vertical and horizontal	.....	Large myopic crescent
33. Goar	1928	Yes	Nasal to center	No	Chocolate brown	Yes	Oval	No	Yes	6/6, 6/6	.....
34. Friedman	1929	Yes	Center	No	Chocolate brown	Yes	Oval	Yes	Oblique 15	6/6, 6/6	.....
35. Korobova	1929	Yes	Center	Yes	Brown	Yes	Oval	No	Yes	.....	.....
36. Edgerton	1929	Yes	Center	No	Golden brown	Yes	Oval	No	Yes	6/5, 6/5	None
37. Griscom	1929	Yes	Center	No	Brown	Yes	Oval	No	Yes	5/40, 5/40	None

Spindle Reported in the Literature

Acquired Defects	Size of Spindle	Color of Iris	Refraction	Degree of Error	Complaint	Pigment Densest at	Axis of Astigmatism	Age	Sex	Glasses Worn Previously	Fields	Wassermann Reaction
Fibrous opacities	4 by 3 mm.	Brown	Myope	—3	.....	Center	Oblique	45	Female	.....	.....	.....
Posterior staphyloma	3.5 by 4.5 mm.	Brown	Myope	—9	.....	Center	Oblique	50	Female	.....	.....	.....
Fibrous opacities	3.5 by 4.5 mm.	Brown	Myope	—1	.....	Center	Oblique	65	Female	.....	.....	.....
Incipient cataract; fibrous opacities; myopic fundus	3 by 4 mm.	Brown	Myope	—15	Poor vision	Center	Oblique	60	Male	.....	.....	.....
.....	.....	Light	Myope	.....	.....	Center	Oblique	22	Female	.....	Nasal field contracted	.....
Bilateral optic atrophy	.....	Steel gray	Hyperope	.....	Poor vision	Border	Oblique 10	63	Female	.....	White and color fields contracted	.....
.....	2.5 by 1 mm.	Dark brown	Hyperope	.....	Dull pain in eyes	Center	Oblique 30-180	32	Female	8 years	.....	.....
.....	3 by 1 mm.	Bluish-gray	Myope	Low	Symptoms of eyestrain	Center	Oblique 120-60	32	Female	10 years	.....	.....
.....	3 by 1 mm.	Brown	Myope	Moderate	Poor vision	Center	Oblique 65-15	40	Female	25 years	.....	.....
Nystagmus on movement to left	2.5 by 3 mm.	Brown	Myope	Low	.....	Center	Oblique 180-97.5	25	Male	.....	.....	.....
.....	.....	Brown	Myope	.....	.....	Center	.....	..	Female	.....	.....	.....
.....	.....	Brown	Myope	.....	.....	Center	.....	..	Female	.....	.....	.....
.....	.....	Brown	Myope	.....	.....	Center	.....	..	Female	.....	.....	.....
.....	.....	Brown	Myope	.....	.....	Center	.....	..	Female	.....	.....	.....
.....	.....	Brown	Myope	.....	.....	Center	.....	..	Female	.....	.....	.....
.....	.....	Brown	Myope	.....	.....	Center	.....	..	Female	.....	.....	.....
.....	.....	Brown	Myope	.....	.....	Center	.....	..	.....	.....	.....	.....
.....	.....	Brown	Myope	High	.....	Center	.....	56	Female	.....	.....	.....
.....	.....	Brown	Myope	High	.....	Center	.....	60	Female	.....	.....	.....
Acute glaucoma	.....	Brown	Myope	—15	Glaucoma	Center	.....	60	Female	Yes	.....	.....
.....	.....	.....	Myope	.....	.....	Center	.....	35	.....	.....	.....	.....
.....	.....	.....	Myope	.....	.....	Center	.....	40	.....	.....	.....	.....
.....	.....	.....	Myope	.....	.....	Center	.....	50	.....	.....	.....	.....
.....	.....	Brown	Myope	.....	.....	Center	.....	60	.....	.....	.....	.....
.....	.....	Brown	.....	High	.....	Center	.....	60	Male	.....	Contracted above and below	.....
.....	.....	Gray	Myope	.....	.....	Center	.....	..	Female	.....	.....	.....
Choroidal atrophy	1.5 by 3 mm.	.....	.....	.....	.....	Center	.....	30	Female	.....	.....	.....
.....	.....	.....	.....	.....	.....	Center	.....	31	Female	.....	.....	.....
.....	.....	.....	Hyperope	.....	.....	Center	.....	54	Female	.....	.....	.....
.....	.....	.....	.....	.....	.....	Center	.....	..	Female	.....	.....	.....
.....	.....	.....	.....	.....	.....	Center	.....	..	Female	.....	.....	.....
Cataract	.....	.....	Myope	—14, —10	Cataract	Center	.....	63	Female	.....	.....	.....
.....	.....	Bluish-gray	Myope	.....	Poor vision	Center	Oblique 50	40	Male	.....	.....	.....
.....	⅔ corneal diameter	Bluish-gray	Myope	.....	.....	Center	.....	32	Male	.....	Normal	.....
Uveitis; cataract	.....	.....	Myope	.....	.....	Center	.....	50	Male	.....	.....	.....
None	3.5 by 1 m.m.	Brown	Hyperope	.....	Relief from pteryglum	Center	.....	38	Male	No	Normal	Negative
Fibrous opacities; choroiditis	3 by 1 mm.	Brown	Myope	High	Pain in eyes	Center	.....	36	Female	Yes	.....	Positive

the spindle and became more disseminated toward the periphery. The remainder of the cornea was clear. There was no evidence of ocular inflammation, nor were there any remnants of pupillary membrane.

Butler stated that the pigment rings seen in a case demonstrated by Professor Vogt in Zurich contained a central dot. At no other place in the literature could I find a similar reference, and apparently it is the only case of such a condition reported. It did not exist in the case under study.

It is possible that some cases have been inaccurately reported as Krukenberg's spindle, and the condition has probably been overlooked in a great many instances. Good illumination is essential for the correct diagnosis.

Moeschler stated that examination of 395 eyes with the slit-lamp disclosed pigmentation in 47 per cent. Of this number, 6 per cent showed a heavier deposit of pigment, and he was inclined to think that some of the more dense deposits were possibly a transition to the Krukenberg type of spindle.

Weinkauff reported a case of what he thought was congenital pigmentation of the cornea, but the patient gave a history of syphilis of two years' standing. There were vitreous opacities and spots of choroiditis. The pigment granules were on the posterior surface of the cornea and ran in a vertical direction, but this case was probably not one of the Krukenberg type. Weinkauff had not examined the patient before the syphilitic infection, and he could not decide whether or not the pigmentation was due to syphilis.

I believe that some of the more recent cases reported with a history of ocular inflammation might well be classed with the last mentioned type reported by Weinkauff. Most observers have regarded these pigment spindles as congenital. There was no history of ocular inflammation in the great majority of the cases reported, and the general characteristics of the pigment in each case did not point to an acquired condition. The corneas were transparent and smooth and showed no evidence of former inflammation or vascularization. Pathologic conditions elsewhere in the eye and intra-ocular growths or wounds that might produce corneal pigmentation could be eliminated.

There are, however, a number of other causes of corneal pigmentation, and even though some of them have points of similarity to the Krukenberg type, they should not be confused with it. This has been observed in hemorrhage between the layers of the cornea in cases of iridocyclitis and following operations in the anterior chamber.

Buchanan stated that blood pigment of the cornea is due to recurrent hemorrhages in the anterior chamber.

Wells reported a case of corneal pigmentation which was due to hemorrhage into the anterior chamber following sclerotomy. The color

was green at first and later turned gray. Hemorrhage is a common cause of acquired melanosis, but sections reveal the pigment to be in the anterior part of the corneal stroma. Trauma is another cause of pigmentation. Chemicals, such as aniline dyes, silver nitrate, etc., are responsible for many cases of corneal pigmentation. Senn referred to eighteen cases occurring in workers in aniline dyes, in which the cornea was sepia brown.

Pigmentation has been noted on the posterior surface of the cornea following operations, in which case the pigment was found on the scars. Oeller observed this and thought that it came from the iris, and that its deposition was due to the direction of the flow of the lymph current toward the wound that it healed.

Discoloration of the cornea was observed by Salus in a patient with multiple sclerosis. It assumed a ring form and was thickest at the limbus. Other cases, similar to this, have been reported in patients suffering with multiple sclerosis, and Salus referred to this condition as a cause. Fleischer reported three similar cases and concluded that the discoloration is a symptom of multiple sclerosis. He regarded the corneal staining as hemochromatosis. These are, no doubt, cases of Fleischer-Kayser ring. He published an article that dealt with this type of pigment, which he called a peripheral brownish-green corneal coloration. He stated that it was found particularly in cirrhosis of the liver, pancreatic disease and insular sclerosis of the brain and cord. He stated that this pigment was hematochromic in origin. The deposit was in the conjunctiva and Bowman's membrane, rather than in the corneal stroma. This was proved by section. Later investigators, however, designated this peripheral pigmentation as degenerative changes in the lenticular ganglion. Holloway and DeLong have reported two cases. In their second case, slit-lamp examination revealed that the pigmentation involved Descemet's membrane. It was brown, and the greenish tinge was thought to be due to the underlying blue iris. Hall, Barnes and Hurst, Greenfield, Poynton and Walshe, Archanguelsky, Kestenbaum, Hessberg, Twelmeyer, Cords and Vogt reported similar cases.

It is possible, however, and equally probable that congenital pigmentation of the cornea has the same origin as persistent pupillary membrane or is closely related. The color of the spindle is the same as that of the iris pigment, which is always brown whether the iris is blue or brown. While it is true that the youngest patient in the cases reported was 22 years of age, this does not eliminate the possibility of a congenital development. There are several cases in the literature that illustrate this union of a congenital corneal pigmentation and persistent pupillary membrane. The membrane may extend from the posterior surface of the cornea to the iris and even on to the lens capsule. Samelson cited such a case, as did Makrocki, Zinn, Vossius,

Wüstefeld and Rumschewitsch, but they did not state that these unusual types of pigmentation, although considered congenital, were of the Krukenberg type. They were inclined to think that persistent pupillary membrane and melanosis of the cornea had the same origin, whether of the Krukenberg type or not, and that the origin was not inflammatory.

Wüstefeld believed that it was probably due to the fact that the pupillary membrane had been completely absorbed and the pigment remains left on the endothelial layer. He cited as an example his own case of persistent pupillary membrane, which showed also a fine, curved brown line of pigmentation on the posterior surface of the cornea. He did not point to this as a case of Krukenberg's spindle.

Brückner stated that if one thinks of the fibers of persistent pupillary membrane as broken apart from the corneal surface and the fibers then becoming absorbed, the condition remains that Krukenberg described as melanosis of the cornea. He regarded the broken fiber stage as a transitional stage to the type of spindle described by Krukenberg.

Ballantyne said that the connection of the iris and pupillary membrane with the cornea and lens appears to be in the nature of a gluing or soldering of the parts. At the points of contact, the endothelium on the posterior surface of the cornea disappears, so that the iris and pupillary membrane come into direct contact with Descemet's membrane which remains intact, and the endothelium can be followed up into the angle between the adherent cornea and the iris, where it abruptly ceases. Along the line of contact, the iris and Descemet's membrane lie in immediate contact; only a few little spaces exist, in which are included a few endothelial cells. Therefore the adhesions between the parts must have formed at a date subsequent to the formation of the anterior chamber, Descemet's membrane and its endothelial layer. Normally the lens and iris lie close to the posterior surface of the cornea until shortly before birth, and yet there are no signs of inflammation of the posterior surface of the cornea, iris and ciliary body. There are two explanations for these adhesions.

Mann stated that associated with the formation of the fetal blood supply is the appearance of the anterior chamber and the differentiation of the substantia propria of the cornea and the mesodermal stroma of the iris. This occurs at from 18 to 20 mm., long before the ectodermal portion of the iris has begun to extend out from the margin of the optic cup. After the separation of the lens vesicle from the surface ectoderm the surrounding mesoderm grows in and fills the space between the lens and the surface. Around the lens this mesoderm is continuous with that in the interior of the optic cup. At or before the 18 to 20 mm. stage, vacuoles begin to form in this mesoderm; these, coalescing, lead to a split which separates the mesoderm between the lens and the

surface ectoderm into two layers. Of these, the superficial one is the thicker, and remaining in contact with the surface ectoderm, it subsequently develops into the substantia propria of the cornea. The deeper and thinner layer remains in contact with the anterior surface of the lens. Its central portion finally disappears, while its peripheral portion, which from the beginning is thicker, remains as the superficial layer of the iris stroma peripheral to the lesser circle.

Makrocki believed that in the embryo the undifferentiated mesoblast lying between the lens and the ectoderm splits, one layer going to the mesoblastic part (substantia propria), and the posterior forming the stroma of the iris and pupillary membrane. The cleft between becomes the future anterior chamber. If this separation is interrupted at certain points, owing to a defect in development, the pupillary membrane remains adherent to the posterior surface of the cornea.

Maschimo cited a case of right-sided corneal pigmentation and indicated the cause as defects in Descemet's membrane. In the case described, a defective constriction of the lens vesicle may have played a part. He did not state that this case was of the Krukenberg type.

Waardenberg reported several cases of melanosis of the cornea. He regarded the pigmentation as merely a displacement of pigment, not as an actual melanosis. No instance of cancer is known to have developed from such an eye. Waardenberg saw no evidence of a familial or hereditary character of the anomaly, and it did not interfere with vision. The symmetrical pigmentation in one or both eyes was in the form of dark brown stripes, horizontal or vertical. The iris was gray or brown. This type of pigmentation was probably not spindle-shaped, or the author would have mentioned its oval tendency. But he regarded it as a developmental defect on the posterior layers of the cornea, and he considered it to be misplaced pigment rather than an excess of pigment.

Wintersteiner believed that the adhesions were due to keratitis as the result of intra-uterine inflammation and adhesions taking place between the posterior surface of the cornea and persistent pupillary remains. He claimed that the iris contains no pigment at the time in fetal life when the pupillary membrane is contiguous with the cornea, and that therefore the pigment left from the pupillary membrane could not fuse with the cornea and so could not be a developmental defect.

Fuchs considered melanosis corneae as a congenital pigmentation of the cornea and stated that when the pigment is seated in the endothelial layer, like the pigment remains found on the lens, it represents the remains of a pupillary membrane.

Yamaguchi said that primary melanosis of the cornea is very rare. He assumed a metabolic formation of pigment from the albuminoid bodies of the cells. The corneal epithelium acquired the specific faculty

of the cells to produce and accumulate pigment which is congenitally inherent in the epithelia of the limbus.

Mans believed that congenital corneal pigmentation is due to a defect in the endothelium and in Descemet's membrane. If the ectoderm is injured, then the lens formations and the cornea formation will be affected.

Goar stated, as an explanation of the morphology of the spindle, that in fetal life when the mesenchyme grows inward to form the primitive cornea it may fuse above and below, leaving at some stage a vertical slit which later becomes the pupil, closed by a connective tissue membrane which, as Stock stated, possesses the power of pigment formation.

Greef reported five cases of slitlike pupils in man, but he gave as an explanation that this unusual condition is a retrogression of the eyes to a form that was probably common in early stages of the evolution of man. He regarded this as a coloboma of the iris.

Collins stated that before the third month of fetal life the posterior layer of the cornea is a hyaline membrane and behind it are two layers of cells. The external layer is the endothelial layer, and the internal one becomes the anterior sheath of the lens. Descemet's membrane develops early in fetal life and is deposited from the endothelial cells.

Lewis said that melanosis of the cornea, when an aberration of development, is characterized by pigmentation of the deepest layers of the middle portion of the cornea and is usually symmetrical in both eyes. In the acquired form, it may develop when the anterior chamber has been empty for a long time and the iris remains in contact with the cornea. The iridic pigment is then taken up by the cornea, on the posterior surface of which it has been deposited.

Kollicker and Hertwig stated that after the lens vesicle closes, the mesoderm thickens and later divides into two layers. The outer layer becomes the cornea and the inner the pupillary membrane. The edges of the pupillary membrane develop into iris stroma. As a result of this, the anterior chamber is formed. Parsons stated that this cleavage begins as early as the sixth week.

Collins and Mayou stated that the lens vesicle becomes separated from the surface epiblast by the growth of mesoblastic cells between them. From the mesoblast are gradually differentiated the substantia propria, Descemet's membrane and its lining epithelium. An atypical development of the mesoblast intruding between the surface epithelium and the lens vesicle is a cause of many anomalies of the cornea, and an excessive deposition of pigment results in melanosis of the cornea.

Collins stated that before the iris is formed, there exists between the posterior surface of the cornea and the anterior capsule of the lens the anterior portion of the fibrovascular sheath. This receives its blood

supply partly from the ciliary arteries and partly from those in the posterior fibrovascular sheath which is prolonged forward and around the sides of the lens to join it. The anterior fibrovascular sheath is developed by a differentiation and vascularization of the posterior part of the mesoblast which grows in to separate the cuticular epiblast from the lens. The more anterior portion of this mesoblast develops into the substantia propria of the cornea and Descemet's membrane. At one time, then, the cornea, anterior fibrovascular sheath and lens lie in close contact with one another. Until the separation of the anterior fibrovascular sheath from the back of Descemet's membrane occurs, there is not even a potential anterior chamber. The iris, as it grows out of the ciliary body, consists of some mesoblastic cells and of the two layers of the secondary optic vesicle. The posterior is only slightly pigmented. It has to insinuate itself between the cornea and the anterior fibrovascular sheath on the one side and the lens on the other, pushing the prolongation from the posterior fibrovascular sheath in front of it. The anterior fibrovascular sheath, when the iris has grown in, becomes raised from the surface of the lens capsule and forms the pupillary membrane, which later disappears as far as the small circle or corona of the iris.

After the development of the secondary optic vesicle, mesodermal cells grow between the ectoderm and the lens, which comes from it, as do also the iris and the cornea. This embryonal tissue later divides into separate layers, and if part of the pupillary membrane does not develop normally, it may persist in the pupillary area. This would lead to an abnormal development of the eye and would result in tissues that could not atrophy and disappear. The pigment granules could thus be left on the posterior surface of the cornea, as the cornea and iris lie in apposition a long time before the anterior chamber is formed.

If the primitive cornea and vascular membrane (pupillary membrane) had failed to be completely separated, one would expect to find later a continuity between them. Descemet's membrane also might show defects, where cleavage had failed to occur. But one finds a normal cornea and iris, Descemet's membrane intact and the appearances suggestive of adhesions of the iris and the posterior cornea, with the remaining pigment on and in the endothelial layer. It is probable that at some time in fetal life, while the iris and Descemet's membrane are in contact, there is a disturbance in cleavage and that as a result pigmentation occurs on the cornea, owing to a disturbed relation in the normal development. If it was due to intra-uterine inflammation there would no doubt be some evidence of newly formed tissue and it would hardly be possible that an inflammatory process would undergo complete resolution. Further microscopic observation will be necessary before its

origin is satisfactorily determined. Still, one cannot deny the possibility in some cases that an inflammation may have been the agent in the development of the pigmentation. Even so, it might have been a coincidence. The cases reported with a history of ocular inflammation are too few to conclude that this, rather than a congenital defect, is the determining factor in the causation of the spindle.

The reason for the linear arrangement of pigment on the posterior corneal surface may be due to the circulation of the aqueous. Turk's theory was that the currents in the aqueous were due to a difference in temperature in the anterior chamber, the portion nearer the cornea having a lower temperature, and the cellular elements which circulate in the aqueous being deposited in the center of the cornea. The circulation is more active in the center than in the lateral zones, and due to the irritation thus produced by the pigment, some of it may be deposited there. The center of the corneal surface is farthest from the iris and has a lower temperature than near the limbus.

The pigmentation may be due to the diffusion of pigment from the pupillary membrane, which in fetal life lies against the posterior surface of the cornea for a certain length of time. It is still a question whether the retention of the pigment is favored by a close union of the two membranes, due to an early rupture of mesenchyme in the true cornea and pupillary membrane, whether it is due to local irritation after formation of the pupillary membrane, or whether it is due to an interruption in absorption of the membrana pupillaris.

COMMENT

I have no new theory to account for the origin of the spindle. In spite of the fact that several of the later cases suggest the theory of inflammation as a cause, I believe that the spindle is of congenital origin because of the symmetry, as practically all of the cases have been bilateral. One case, Friedman's, was reported with persistent pupillary membrane. The pigmentation is always deep in the corneal tissue posterior to Descemet's membrane. There is no evidence of ocular inflammation or trauma, except in one case presented. It is almost always associated with myopia, and there is much yet to be learned about the embryonic pathology of myopia. Five per cent of persons are myopic at birth, according to Fuchs.

Some cases reported revealed various types of congenital as well as acquired conditions, among them being incipient cataracts, vitreous opacities, coloboma of the disk, optic atrophy, acute glaucoma, choroidal atrophy, nystagmus and myopic fundus changes.

The pigmentation does not always occur in brown eyes, as Krukenberg thought, for it has occurred many times in lighter irides.

In the cases reviewed vision seems to be as good as in the eyes without pigmentation, except perhaps in the one case reported by Friedman. The patients were not aware of the fact that they had this melanosis. The spindle was vertical in all cases, except those reported by James and Kraemer. It usually occurred in myopes, but there were several cases reported in which hyperopia existed. All were symmetrical and bilateral except four, which were unilateral. Two of these were reported by Koby, one by James and one by Kraupa. The location of the spindle was central or nearly so in all, and was placed in the deeper layers of the cornea. The size of the spindles varied slightly, ranging from 2.5 by 1 mm. to 4.5 by 3.5 mm. The color of the iris was brown in twenty cases. Seven showed blue or gray irides and in ten cases the color was not mentioned. Twenty-nine patients were myopic in varying degrees. Four patients were hyperopic, and in four the refraction was not stated. In most cases in which the refraction was reported, the axes were oblique. Twenty-four cases were in females, eight in males and in five the sex was not stated. The youngest patient was a man, aged 22, whose case was reported by Thompson and Ballantyne. Thirteen were between the ages of 22 and 45 years. Seven were over 60. The age was not given in ten cases. Griscom's case was the only one reported in a colored person and the only case showing a positive Wassermann reaction.

Dr. C. E. G. Shannon provided the facilities of the Jefferson clinic, without which the work on the case reported would have been impossible; Dr. Alfred Cowan made several slit-lamp studies in this case and Dr. I. S. Tassman gave valuable assistance in translating some of the German articles.

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Obituary

G. C. SAVAGE, M.D.

1854-1930

It is with great regret that the passing of a respected colleague, Dr. Giles Christopher Savage, is chronicled. He died at his home in Nashville, Tenn., on April 8, 1930. Dr. Savage was a native of Mississippi, being born at Rienzi, in 1854. His father was a Baptist minister, and into that faith Dr. Savage was born, lived, worked and died. He studied medicine at Jefferson Medical College, Philadelphia, and after graduation in 1878 practiced for a while at Jackson, Tenn. He then moved to Nashville, where he taught ophthalmology in Vanderbilt University from 1886 to 1911. He was president of the Tennessee State Medical Association, first vice-president of the American Medical Association and Chairman of the Section on Ophthalmology. His contributions to ophthalmology comprise the founding of the *Ophthalmic Record* in 1891, which he edited for many years till it was finally merged in the *American Journal of Ophthalmology,* and two books relating to the ocular muscles, namely, "New Truths in Ophthalmology" and "Ophthalmic Neuro-Myology." He also wrote the chapter on the physiology and pathology of "ocular muscles" in the "American Encyclopedia of Ophthalmology," and was the author of many articles that appeared in various ophthalmic and other journals. Dr. Savage was greatly interested in religious subjects, and was an officer and active member of the Baptist church. He wrote "Time and Place Harmony of the Gospels" and other religious books that had a large circulation.

Dr. Savage impressed his medical associates with his enthusiasm on the subject of the ocular muscles, a subject to which he gave much time and thought. While his confidence in muscle imbalance as a cause of reflex disturbance of all kinds was not shared by his colleagues, it encouraged him to a study of the question from which all have profited.

Dr. Savage was gifted with a splendid speaking voice, an excellent command of language, a keen sense of humor and a temperament that was never ruffled in debate, even when his most cherished ideas were vigorously attacked. He was fair to his confrères and kind and encouraging to younger men, for which he is held in grateful memory.

Dr. Savage was very active in the plans for the centennial meeting of the Tennessee State Medical Association. One week before the meeting he was taken ill with pneumonia, and died just before the opening session was called to order.

E. C. ELLETT.

Correspondence

ANKYLOBLEPHARON FILIFORME ADNATUM

To the Editor:—With reference to the report of a case of ankyloblepharon filiforme adnatum by Drs. H. V. Judge, W. C. Mott and J. A. C. Gabriels, which appeared in the December issue of the ARCHIVES OF OPHTHALMOLOGY (2:702, 1929), may I be permitted to point out that a similar case was recorded in the annual report of this hospital for 1926? The following note is quoted from the report:

"A full-term Hindu female child, aged 5 days, was brought to hospital on Feb. 11, 1926, by the mother because the lids would not open properly. On examination it was found that several narrow bands of tissue held the margins of the lids together, presumably the remains of the tissue union of early intrauterine life. On the left there was only one narrow stretched out bridge at the centre of the lid which looked as if it might break at any time. On the right there were several bands close together towards the middle third of the palpebral aperture. The lids were only capable of partial opening. The condition is very uncommon."

ROBERT E. WRIGHT, M.D., Madras, Spain.
Government Ophthalmic Hospital.

"CORNEAL SPUDS"

To the Editor.—In the April issue of THE ARCHIVES is a short note on "Corneal Spuds" by Dr. Fred J. Walter, of San Diego, Calif., in which he describes three varieties. Number three was devised by me twenty or more years ago and has been on the market for at least fifteen years. It is manufactured by E. B. Meyrowitz of New York and by V. Mueller & Company of Chicago. I described this spud in one of the defunct ophthalmic journals, but which one I cannot recall. I had long felt that most spuds on the market were too large, and a point that I made in the instruction to the manufacturer was that this one should be only 0.5 mm. I am glad Dr. Walter has found this spud to be of practical value.

A. G. BENNETT, M.D., Buffalo.

Notes and News

EDITED BY DR. JOHN HERBERT WAITE

SOCIETY NEWS

Cleveland Ophthalmological Club.—The regular dinner meeting of the club was held on April 15, with Dr. Martin Cohen of New York as the guest of honor. Dr. Cohen spoke about "Syphilitic Affections of the Eye."

New York Association for the Blind.—The association has opened the Lighthouse Clinic for the Prevention of Blindness at 114 East Sixtieth Street, New York. This is an evening clinic to serve the preschool child and the wage earner.

Utah State Medical Association.—The association will meet in Salt Lake City, September 9, 10 and 11.

Ontario Medical Association.—At the Royal York Hotel at Toronto, on May 28, the Ophthalmic Section of the Ontario Medical Association will present the following program: Dr. Fred T. Tooke, "The Ophthalmologist in Consultation"; Dr. F. R. Bonnetto, "Ophthalmic Accidents in Industry"; Dr. Sanford Gifford, "Practical Ophthalmic Procedures," and Dr. Wilbur G. Fraser, "Cataract Extraction with Peripheral Iridectomy." In a symposium on melanotic sarcoma the histories, treatment and end-results in thirty-five cases will be reviewed.

Ophthalmological Society of the United Kingdom.—The society recently celebrated the fiftieth year of its existence. It originated from a small group of distinguished ophthalmic surgeons who met informally at Moorfields. The inaugural meeting was held in February, 1880, at the house of Sir William Bowman in London, and its first ordinary meeting was held on June 23 of that year. The first council of the society included: George Critchett, Jonathan Hutchinson, Hughlings Jackson and Henry Swanzy (vice presidents), Nettleship and Stephen Mackenzie (secretaries) and Gowers, Brudenell Carter, Hulke, Henry Power and Waren Tay (members of the council). Of the 114 original members of the society, 11 survive. Sir George Berry and Mr. J. B. Lawford were present at the congress, and Sir Thomas Barlow, one of the committee that originated the movement, responded to the toast of the society at the jubilee banquet on April 4. A number of distinguished foreign ophthalmic surgeons attended as guests of the society. The Royal College of Surgeons held a reception on April 3. The president of the congress was Mr. Leslie Paton. The forty-nine published volumes of the *Transactions* prove how fully the society has borne out the aspirations of Bowman's inaugural address. Thus, in the first volume appears Waren Tay's description of the disease now known as

amaurotic family idiocy, and Hughlings Jackson's admirable account of optic neuritis in cerebral lesions. The Bowman Lectureship was instituted in 1885 to commemorate the first president, and the first lecturer was Hughlings Jackson. Many men of great distinction succeeded him. This year, Sir Arthur Keith lectured on April 4 on the genius of Sir William Bowman. In 1912 the question arose of amalgamation with the Royal Society of Medicine. The society decided to conduct an annual congress and agreed to leave the monthly meetings to the Section. That arrangement has resulted in building up a strong Section of Ophthalmology in the Royal Society of Medicine; the Ophthalmological Society is stronger than ever, and works in complete harmony with the Section.

British Medical Association.—There will be a meeting of the association in Winnipeg, Aug. 26 to 29, 1930.

GENERAL

Lectures by Dr. Ida C. Mann.—The following is a synopsis of six two-hour lectures on "The Phylogeny and Ontogeny of the Human Eye," to be given in Denver, beginning September 29, and in Chicago, beginning October 13, 1930, by Dr. Ida C. Mann, of London.

1. The scope of the course. Tropism. Light reactions in unicellular organisms. Visual cells. Types of eyes found in invertebrates. Simple and compound eyes.

2. Theories of the derivation of the vertebrate from the simple invertebrate eye. Cerebral eyes versus cutaneous eyes. Early stages in vertebrate embryology. The three germ layers. The derivation of the various parts of the eye from these. Ocular characteristics of fish and amphibia.

3. The eye (development and adult structure) in reptiles, birds and lower mammals. Pineal and parietal eyes.

4. Detailed consideration of human development. Early stages in human embryos. Methods of measuring human embryos. Gross changes of shape of the ocular outgrowth. The development of the lens in detail. Structures derived from neural ectoderm. The vitreous. The zonule.

5. The structures derived from the mesoderm. The fetal and adult blood supply. The choroid and sclera. The muscles and other structures in the orbit. The alteration of optic axis during development.

6. A consideration of the development of the commoner congenital anomalies found in man.

Any one desiring to take the course in Chicago, please communicate with Dr. R. von der Heydt, or Dr. Beulah Cushman, 25 East Washington Street.

Ophthalmic Examination on June 23.—The next examination of the American Board for Ophthalmic Examinations will be held at the Harper Hospital, Detroit, on June 23. Applications should be made to the secretary, Dr. William H. Wilder, 122 S. Michigan Ave., Chicago.

PERSONAL

Dr. Morax, ophthalmologist to the Hospital Lariboisière, has been elected a member of the Academie de Médécine in the surgical section.

Dr. George E. deSchweinitz, professor of ophthalmology in the Graduate School of Medicine at the University of Pennsylvania, has been awarded the Leslie Dana Medal for prevention of blindness, award of 1930. This medal has been given each year since 1925 to the person who performs outstanding work in the prevention of blindness and in the conservation of vision.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Anatomy and Embryology

ORBITAL BRANCHES OF THE SPHENOPALATINE GANGLION AND THE PRESENCE OF SMALL NERVE GANGLIONS IN THEIR COURSE. G. VITALI, Riv. oto-neuro-oft. **6:** 151 (March-April) 1929.

Professor Vitali, director of the anatomic institute of Pisa, has studied this uncertain subject in thirty-five orbits sectioned serially and examined microscopically. It was invariably found that the fibromuscular tissue occupying the inferior orbital fissure receives numerous nerve filaments from the sphenopalatine ganglion. Small nerve ganglions were found on these filaments and on a filament anastomosing with the abducens, as well as in the fibromuscular tissue itself.

Confirmation was obtained of the existence of a nerve filament uniting the sphenopalatine ganglion to the sheath of the optic nerve, admitted by Hirzel and Arnold, denied by Beck. A branch anastomosing with the abducens was found as described by Varrentrapp, but denied by Gross and Hirzel. One and the other, as well as a branchlet anastomosing with the sympathetic plexus of the cavernous sinus, were invariably found when studied in six complete series.

The author did not find a filament going to the ciliary ganglion, as seen by Tiedemann, Arnold and Valentin, but never observed by Varrentrapp; nor did he meet the spheno-ethmoidal branches of Luscka, which many textbook writers admit, but which must be considered, at least, inconstant.

The presence of nerve ganglions and nerve fibers in the fibromuscular tissue of the inferior orbital fissure must be considered as important anatomic data in the interpretation of the functional value of this smooth muscle tissue. It may be adduced to support the belief that Mueller's muscle has a certain importance in the pathogenesis of exophthalmos in morbus Flagiani-Basedow.

Of the various parts of Mueller's muscle, that part which occupies the inferior orbital fissure is capable of causing stasis in the ophthalmic venous system by the compression which it can exercise on the venous channels traversing it, especially on the larger anastomoses joining the inferior ophthalmic vein with the deep branch of the anterior facial vein. This stasis, of course, would be relative and not absolute.

V. R. SYRACUSE.

Congenital Anomalies

CONGENITAL COLOBOMA OF THE UPPER EYELID WITH DERMOIDS OF THE CORNEA. W. D. HORNER and F. C. CORDES, Am. J. Ophth. **12:** 959 (Dec.) 1929.

The authors refer to the rarity of coloboma of the lid and discuss its appearance and extent, the other congenital defects associated with

it and its etiology. Dermoids are also discussed. The case reported occurred in a woman, aged 28. The left upper lid showed a cleft 19 mm. wide and 6.3 mm. deep. Just above the cleft was a mass, evidently a lipoma. There was another beneath the bulbar conjunctiva near the external canthus. The lower limbus was straddled by a dermoid, as was also the temporal limbus, their diameters being 9.5 and 4.7 mm., respectively. These tumors affecting the globe were excised, and a laboratory diagnosis of dermoid was made of those astride the limbus, with aberrant lacrimal gland of the subconjunctival mass.

W. S. REESE.

CASE OF ATYPICAL COLOBOMA ASSOCIATED WITH ABNORMAL RETINAL VESSELS. I. MANN and J. A. ROSS, Brit. J. Ophth. **13**: 608 (Dec.) 1929.

In a previous paper by Mann, macular colobomas were grouped into three classes, pigmented, nonpigmented and macular coloboma associated with abnormality of blood vessels. The various theories advanced to explain these conditions were discussed, and the balance of evidence was thought to be in favor of the occurrence of an antenatal choroiditis as the determining factor.

The present case belongs to the third group. The patient was a man, aged 59. With correction, vision in the right eye equaled 6/6 and in the left eye 6/9. In the left eye the superior temporal vessels were represented by a single threadlike vein. Up and out from the disk, a pearly white cup was seen. Its margin overhung to the lower half of its nasal side, where it was shelving. The cup was deep and from it emerged large vessels which supplied the upper temporal quadrant of the fundus. Between the disk and the cup (or coloboma) the choroidal vessels were easily seen, and the diminutive superior temporal vein anastomosed with them at two points. At the nasal edge of the cup there were groups of faint grayish dots here and there around the disk. The macular area was stippled.

The field showed a normal blind spot and (apparently continuous with it) a scotoma corresponding with the coloboma. Color vision was normal.

The case then is one of note, and, as far as the author knows, unique in that a complete quadrant of the retina is supplied by a group of adventitious vessels appearing in the fundus through a localized defect in the retina and choriocapillaries above and outside the disk. Mann discusses in detail the embryologic considerations, which are here given in abstract. The probable sequence of events appears to be that a patch of choroiditis (or choroidal disturbance of unknown etiology) probably occurred during the fourth month, involving the choriocapillaries and external choroidal layer but not necessarily destroying the intermediate layer. The sclera might become slightly ectatic and the overlying retina be also involved to a certain extent. The pigment layer and Bruch's membrane would be destroyed. The rods and cones would probably suffer, but the inner layers, especially the nerve fiber layers, would not necessarily be completely destroyed. This would explain the absence of a sector defect in the field. The disturbance of Bruch's membrane would make it possible for new arteries to invade the overlying retina.

The development of the upper temporal branch of the central artery of the retina would be inhibited since its area of supply would be already vascularized by the abnormal vessels. The abnormal artery of supply is presumably one of the short posterior ciliaries.

W. ZENTMAYER.

Refraction and Accommodation

CHANGES OF THE REFRACTION CAUSED BY CONTUSION OF THE EYE. O. HUBER, Klin. Monatsbl. f. Augenh. **83:** 523 (Oct.-Nov.) 1929.

Traumatic myopia of a transient, as well as permanent, type has been observed frequently. The author reports two cases of permanent traumatic hyperopia. The first was in a girl, aged 20, whose eye was struck by a piece of kindling while she was splitting wood. Hyphema, filling the entire anterior chamber, was absorbed within twelve days, leaving the pupil perpendicularly oval, slightly dilated and void of reaction. Incidentally, the patient discovered that she could see with her injured eye at a distance without a glass. The refraction had changed from 2.75 myopic astigmatism (two years previous to the injury) to one half of 1 diopter of hyperopia with 1 diopter of astigmatism, accommodation being perfect. The same condition still existed thirteen months later, the pupil being irregular and showing no reaction to light.

The second case was that of a young man, aged 17, who wore a concave lens of 7.5 diopters for his right and one of 7 for his left eye. His right eye was struck during an automobile accident, but gave no evidence of injury. Examination of both eyes under atropine showed 5 diopters of myopia in the right eye and 7, as before, in the left, a decrease of 2.5 diopters in the injured eye. Excluding changes of the zonula and spasm of the accommodation, the author thinks that elongation of the axis is the cause of the phenomenon in both cases. Shifting of the lens cannot be considered as the cause, for a displacement of 5 mm. would be required to bring about a change of the refraction of 3 diopters.

K. L. STOLL.

Methods of Examination

A NEW METHOD OF DETERMINING THE LOCATION OF LESIONS IN THE EYEGROUND: ITS APPLICATION TO GONIN'S METHOD OF TREATMENT FOR RETINAL DETACHMENT. K. LINDNER, Arch. f. Ophth. **123:** 234 (Dec.) 1929.

Lesions in the eyeground can be localized exactly by having the patient fix on the center of a perimeter arc while the ophthalmoscope is led along the arc until the lesions appear in the center of its field; or the ophthalmoscope is put in the center of the arc and the patient's eye is guided along a fixation mark on the arc until the lesions appear in the center of the field of the ophthalmoscope. The author adapted the Zeiss Gullstrand ophthalmoscope for the latter purpose and so determined the angle which the principal ray going to the lesion subtends with the visual axis. This angle allows an approximate cal-

culation of the distance of the lesion from the corresponding part of the limbus. Furthermore, Lindner determined the meridional plane in which the lesions were located. These two measurements led to the possibility of finding the projection of the lesion on the external surface of the sclera. In cases of retinal detachment the holes, tears or ruptures that are nearly always found can be localized with this method. One of the errors made when the method described is used is that the tear is projected on the sclera in the direction of its principal ray. This location, however, may not be the actual place of the tear because of the manifold dislocations that the detached retina undergoes. By measuring the refraction of the region of the tear, it seems possible to eliminate this error also.

In order to find the point on the surface of the eyeball which corresponds to the localized lesion, Lindner used a small model which consists of a metal ring and which is shaped like the peripheral part of a contact glass. The model carries an arm fixed by a screw in the center of the ring. This arm is given the right length to indicate the place of the lesion on the surface of the eyeball and is fixed in the proper meridional plane. The whole model is tied on the cornea by four sutures which are led through the sclera near the limbus. Where the arm of the model ends, a small mark is made on the sclera with the cautery. Then the model is taken off and electrocautery performed. Lindner leaves the hot cautery in the subretinal space for from ten to fifteen seconds, sometimes as long as twenty seconds.

Lindner used the method described in eleven cases. In ten of these the retinal tear was reached by the first cautery. In seven cases the retina became and remained reattached (at the time of the publication of the paper the longest period of observation after the reattachment was eight months, the shortest, three weeks). Lindner believes that Gonin's method for retinal detachment is one of the greatest accomplishments of modern ophthalmology.

P. KRONFELD.

LOCALIZATION INSIDE THE EYE. M. SALZMANN, Arch. f. Ophth. **123:** 252 (Dec.) 1929.

Following Lindner's suggestion that an accurate localization of the retinal tears in cases of detachment should be attempted, Salzmann has worked out a fairly simple but accurate way of determining the three coordinates of lesions in the eyeground. He lets the patient's eye fix on the center of a perimeter arc and determines the angle under which an ophthalmoscope must throw its light on the eyeground in order to get the lesion in the very center of its field. This angle is called the "Visierwinkel." Then the meridional plane in which the lesion lies is determined. So far Salzmann's procedure is not different from Lindner's method. Only the way in which the measurements are transposed into actual lengths and degrees on the eyeball is different, and in Salzmann's method this is more accurate. Unfortunately, he does not say how big an error is made if Lindner's procedure is carried out. That would be important considering the fact that Gonin, with his rough method of localization, had excellent results in the treatment for retinal detachments.

P. KRONFELD.

ECCENTRIC MYDRIASIS PRODUCED TO DETERMINE THE INDICATION FOR AN OPTIC IRIDECTOMY. M. VANNAS, Klin. Monatsbl. f. Augenh. **83:** 476 (Oct.-Nov.) 1929.

With due credit to the methods of Meller and Gradle and with mention of the use of the stenopeic disk, the author refers to his experiments with diverse mydriatics. Atropine and homatropine produce dilatations involving the entire pupil at once, if injected into any place in the conjunctiva adjoining the limbus. Experiments made with a solution of spurarenin 1 : 1,000 produced partial dilatations within from three to six minutes and of a duration of from two to six minutes, according to the dosage. Glaucosan (an epinephrine substitute) twenty times stronger than the standard suprarenin, may be used on a cotton-tipped probe; it will yield a partial mydriasis if rubbed for from fifteen to thirty seconds with gentle pressure into any desired spot of the limbus, after the use of a nonirritating local anesthetic. The vision can be tested, while the partial mydriasis lasts. Doses of different strength allow the study of the prospective effects of iridectomies of different size.

K. L. STOLL.

General Pathology

HISTOPATHOLOGY OF THE OPTIC NERVE IN ARTERIO-SCLEROSIS AND SENILE DEMENTIA. A. VON STIEF, Ztschr. f. Augenh. **70:** 41 (Dec.) 1929.

It was noted by Stief that arteriosclerosis of the nutrient vessels of the optic nerve did not always parallel that of the surrounding vascular bed. Often the internal carotid, the ophthalmic and several of the small branches of both revealed considerable sclerosis, even thrombosis; yet the small vessels of the optic nerve and its nerve tissue sheaths were not involved. The reverse of this picture was often seen. Generally, if the larger vessels on one side were sclerosed, the vessels on the opposite side were likewise sclerosed. Angiospasm usually preceded definite signs of sclerosis of the vascular bed. Blindness was probably due to pressure atrophy of the vessels on the nerve in the optic foramen or sclerosis of the small nutrient vessels of the nerve and its sheaths. A study of the optic nerve, chiasma and tract of ten cases of senile dementia revealed merely senile changes in the tissue. In two instances a peripheral degeneration similar to that described by Sven Ingvar in his cases of Argyll Robertson pupil was noted. Stief views this observation as a challenge to Sven Ingvar's assumption that marginal atrophy of the nerve is responsible for the lack of reaction of the pupil to light. He further notes that in his cases the pupils did react to light.

A. M. YUDKIN.

Experimental Pathology

THE RELATION BETWEEN THE SPINAL FLUID PRESSURE AND INTRAOCULAR PRESSURE. K. NOISZEWSKI, Arch. d'opht. **46:** 429 (Aug.) 1929.

The author presents two photographs of longitudinal sections through the disk of the eye of dogs: a normal dog and one that had undergone craniotomy some months previously. A distinct depression of the normal elevation of the disk was noted in the animal on which

operation was performed. On this basis the author suggests that simple glaucoma may be the result of a lowered intracranial pressure. Conversely, he points out that there are cases of increased intra-ocular pressure without excavation of the nerve head. He suggests that the intracranial pressure in these cases is high.

S. B. MARLOW.

EXPERIMENTAL SUPRANUCLEAR CATARACT AND NUCLEAR SCLEROSIS. H. GOLDMANN, Klin. Monatsbl. f. Augenh. **83**: 433 (Oct.-Nov.) 1929.

Toxic lesions of the lens begin in the layers adjoining the capsule, e. g., the naphthalene cataract in rabbits, the thallium cataract in rats, the cataract due to tetany in dogs and the diabetic cataract in young persons. This experience leads to the belief that the superficial layers solely are affected by severe toxic actions of short duration on the lens. Goldmann succeeded in obtaining deep-seated chronic changes, not noticeable in the superficial layers, in the lens in rats. These were fed by tube with 0.1 Gm. of naphthalene in 150 Gm. of oil daily for from eight to twelve days. No visible superficial changes appeared, but an opaque layer developed between the capsule and the nucleus, reaching gradually its maximum after from two to three weeks. Complete cataracts were never observed. These opacities were located closer to the center of the lens in the young, than in adult, animals. Experiments were discontinued for some time; their renewal by again feeding naphthalene led to the development of a new and deep-seated opaque layer, adjacent to the former. These lenses had a hard nucleus, of a greenish-brown hue, surrounded by clear cortex. Similar experiments on rabbits failed to produce deep-seated opacities.

K. L. STOLL.

PATHOLOGIC ANATOMY OF THE EXPERIMENTAL TETANY CATARACTS IN DOGS. A. VON PELLATHY, Klin. Monatsbl. f. Augenh. **83**: 438 (Oct.-Nov.) 1929.

Tetany cataracts, according to Siegrist, have been found after extirpation of goiters in cases in which the parathyroids were removed or damaged. Experiments of Siegrist and Goldmann on dogs the parathyroids of which had been removed proved that the opacities of the lens were limited to the subcapsular portion and that this is a direct sequela to the tetanic attacks. These subcapsular opacities were forced to become parts of the deeper layers in the physiologic development of the new lens fibers. From four to six opaque zones were thus observed by the author by means of the slit-lamp and also under the microscope. The epithelium of the capsule showed signs of degeneration, consisting in changes of the form of its cells and occasional necrosis of the cells and their nuclei. The lens fibers carried symptoms of degeneration and vacuoles filled with either myelin or albumin or some other homogeneous substance. These formations, small and fine in the cortical layers close to the surface, became larger and coarser and confluent in the deeper layers. Detritus was found in the larger vacuoles and in the spaces formed by pathologic detachment of the epithelium from the capsule. None of these changes was noticed in the nucleus of the lenses. The ciliary epithelium was free from pathologic changes.

K. L. STOLL.

Lids

TREATMENT OF BLEPHARITIS WITH BRILLIANT GREEN. N. I. MEDVEDEFF and N. Z. EUFFA, Russk. oft. j. **10:** 723 (Dec.) 1929.

The authors treated forty-four patients with a 1 per cent brilliant green solution (an aniline dye of triphenyl-methane group). A 1 per cent brilliant green solution in 50 per cent alcohol was used on twenty-nine patients; on ten patients an aqueous solution of the dye was used, and for five patients one eye was treated with an aqueous solution and the other eye with an alcoholic solution of brilliant green. All patients suffered with marked blepharitis squamosa or ulcerous blepharitis for several years. The usual treatment including correction of refraction errors gave no improvement.

The treatment consisted of daily swabbing of the margin of the lid with a 1 per cent solution of brilliant green. The scales were carefully removed before each application, and precautions were taken to prevent the solution from getting into the conjunctival sac. Patients with blepharitis squamosa received about sixteen applications and those with ulcerous blepharitis about eleven applications.

There was a rapid and marked improvement in all cases, first of the subjective symptoms and then of the objective. The scales disappeared in from five to six days, and the ulcers healed in from seven to ten days. This improvement, according to the authors, is due to the bactericidal action of the dye.

Medvedeff's and Euffa's conclusions are:

1. The usual methods of treatment of recurring chronic blepharitis are unsatisfactory.

2. Autovaccination by the Besredke method is very valuable, but too tedious and too expensive.

3. Alcoholic solutions of brilliant green give a marked improvement in all cases, and in many cases lead to restitutio ad integrum.

4. Recurrence of blepharitis after treatment with brilliant green is not rare, but the cases yield readily to repeated treatment.

5. A 1 per cent solution of brilliant green in 50 per cent alcohol should be at hand in every ophthalmologist's office or clinic.

O. SITCHEVSKA.

Conjunctiva

PSEUDOMEMBRANOUS CONJUNCTIVITIS. G. TESSIER, Ann. di ottal. e clin. ocul. **57:** 494 (June-July) 1929.

The author insists on the value of bacteriologic examinations in cases of conjunctivitis. In the pseudomembranous form a culture is especially important to determine the presence or absence of *B. diphtheriae*. The clinical differentiation between a true diphtheritic membrane and the more superficial forms is of no diagnostic value, since both represent varying degrees of the same reaction. All of the pathogenic bacteria of the conjunctiva can, under certain conditions, cause such membranes.

Tessier reports fifty cases of pseudomembranous conjunctivitis observed since 1923. Fifteen cases occurred with measles and five with

scarlet fever. Three patients were subjects of hereditary syphilis, two were rachitic, three had chronic blepharitis, two had had a previous chronic conjunctivitis and three had pharyngitis which was diphtheritic in two cases. In seventeen cases there was no apparent predisposing cause. Most were children from 1 to 4 years of age. He believes that a poor nutritional state or a lack of proper immunologic reaction was a determining factor in most cases.

Bacteriologic studies showed the following organisms: *B. diphtheriae* alone in four cases, three with corneal complications; pneumococcus in seven, three with corneal complications; streptococcus in fifteen, eleven with corneal complications; *B. subtilis* in one; *B. diphtheriae* and pneumococci in two, one with corneal complications; *B. diphtheriae* and *B. xerosis* in two, one with corneal complication; *B. diphtheriae* and streptococcus in one, with both corneas involved; *B. diphtheriae* with both streptococcus and staphylococcus in one, with both corneas involved; pneumococcus and streptococcus in three, with one corneal involvement; *Streptococcus* and *Staphylococcus aureus* in five, with corneal involvements in three; *Streptococcus* and *Staphylococcus albus* in six, with corneal involvements in two; Koch-Weeks bacillus in two and gonococcus in one, the last three being without corneal involvement. In determining the presence of *B. diphtheriae,* the characteristic granules were looked for, and in two cases injections were made into guinea-pigs. *B. diphtheriae* showed the greatest tendency to involve the cornea, with the streptococcus and pneumococcus next in order.

In cases due to *B. diphtheriae,* the use of large doses of antidiphtheritic serum (from 70,000 to 80,000 units daily) was apparently superior to the use of smaller doses (10,000 units), as shown by the incidence and severity of corneal complications. The serum was of little effect when *B. diphtheriae* were associated with streptococci or pneumococci.

A large number of patients with cases due to other organisms were given antidiphtheritic serum for its nonspecific effect. Neither large nor small doses were shown to have any definite effect on the corneal complications. Oral and local administration were at least as effective, from which the author concludes that such nonspecific treatment, in this condition, is of little or no benefit.

S. R. GIFFORD.

TREATMENT OF TRACHOMA BY ANGELUCCI'S TRACHOLYSINE. DINULESCU, Cluj. med., 1929, no. 6.

Into the composition of Angelucci's preparation, called tracholysine, enter physiologic solution of sodium chloride, 0.3 per cent; sodium sulphate, 0.05 per cent; calcium glycerophosphate, 0.5 per cent; sodium nucleinate, 0.5 per cent; procaine hydrochloride, 1 per cent; phenol, 1 per cent; distilled water, 100 Gm. The author has treated several patients with trachoma with this preparation for five months, by repeatedly giving subconjunctival injections of 1 cc. of this solution within each three days. During the treatment he observed a beneficial effect on the trachomatous process, but after having interrupted the injections the prior condition of the eyes soon reappeared. He feels that this preparation is not to be judged so favorably as chaulmoogra oil.

N. BLATT.

Trachoma

THE TREATMENT OF TRACHOMA: STAGE 1. (MACCALLUM'S CLASSIFICATION WITH CARBON DIOXIDE SNOW.) Research Dept., Giza Mem. Ophth. Lab., Cairo, 3d Ann. Rep., p. 57.

Stage 1 is that in which the tarsal conjunctiva is seen to be mildly congested and in which there are tiny, yellowish-gray, avascular areas shining through. Twenty-five children suffering from trachoma in this stage were treated; the right eye was treated with carbon dioxide snow carefully applied to the area of conjunctiva bearing follicles, in such a way as to produce immediately a thin milky film of necrosis. The conjunctiva of the left eye was rubbed daily with a stick of copper sulphate.

The results have been distinctly favorable to sulphate of copper. More satisfactory cicatrization and smoother lids have been obtained in a much shorter time with it. In some cases after from three to four weeks of daily treatment with copper sulphate almost complete cicatrization has been obtained. Following the application of the snow, the follicles apparently disappeared rapidly, but a form of conjunctivitis was produced which seemed as difficult to treat as the original form.

W. ZENTMAYER.

Lens

RÔLE OF CALCIUM IN SENILE CATARACT. DOROTHY ROSE ADAMS, Biochem. J. 23: 902, 1929.

An original investigation of the author showed that:

"In patients with senile cataract the serum calcium is appreciably higher than normal.

There is no age variation in the serum calcium either of normal subjects or of those with senile cataract.

Calcium salts acting on fresh ox lenses do not cause opacity except in unphysiological concentrations, nor do they act as sensitizers in the production of opacity by ultraviolet radiation.

Dialyzed solutions of lens proteins alpha and beta crystallin exhibit a certain sensitivity to solutions of calcium salts.

The experimental production of a persistently raised blood calcium in rabbits does not cause cataract, nor accelerate the development of naphthalene cataract, although it causes a corresponding rise of calcium in the aqueous humor.

There is no age variation in the calcium content of normal or of cataractous human lenses.

The calcium content of cataractous lenses is much greater than that of normal lenses, while the potassium content is considerably less.

Individual cataractous lenses show a wide variation in calcium content, which may be attributed to an erratic local deposition of calcium, for which there is also some histological evidence.

Although there is an increase of calcium in the blood of patients with senile cataract, and an abnormal deposition of calcium in the lens, it cannot as yet be proved conclusively that the increase in calcium is a primary factor in the actual production of opacity in the lens."

I. S. TASSMAN.

SENILE LESIONS OF THE ANTERIOR CAPSULE OF THE LENS AND OF THE PUPILLARY MARGIN. TRANTAS, Arch. d'opht. **46**: 482 (Aug.) 1929.

The author first reviews Vogt's description of the lesions he described in 1925 in a few cases of chronic glaucoma. He mentions the other cases that have been reported. He then reports the results of the careful slit-lamp examination of 125 male and 112 female patients between the ages of 55 and 80, who came to his clinic for various reasons. Two hundred and fourteen eyes in men and 221 eyes in women were examined. He encountered the changes described by Vogt in thirty-six cases, or 15 per cent. He found six other cases on reexamining some old patients. Chronic glaucoma was present in only fourteen, or 33 per cent, as contrasted to Vogt's 78 per cent. In addition, advanced opacities in the lens were found in thirty cases. Arteriosclerotic changes in the retinal vessels and a variety of other lesions of a degenerative nature were seen in this group of cases. These observations point to the conclusion that these changes are not dependent on glaucoma but are senile. On the contrary, the observation of some similar conditions suggests that glaucoma may follow lesions of this type. Trantas has observed a number of cases in which grayish flocculi have been attached to the cornea, later to fall into the filtration angle and block the canal of Schlemm. Great care must be used in looking for this lesion, as it easily escapes notice.

S. B. MARLOW.

THE INDICATIONS FOR CATARACT OPERATIONS. ADALBERT FUCHS, Cluj. med., 1929, no. 6.

Good light perception and exact light projection give one security prior to the cataract operation that the retina and optic nerve of the cataractous eye are intact. There are, however, many lesions of the eyeground which one cannot detect by ophthalmoscopy until cataract extraction has been done. There are also exceptions regarding light perception and light projection. In a case of hypermature cataract the effect of the operation on vision may be satisfactory, although light projection may exhibit deficiencies; in such cases, the light is often exclusively located on the temporal side. The same may occur in cases of membranous and traumatic cataracts, especially when the capsule is covered with much pigment. On the other hand, in cases of cataract there may be hyposensibility of the retina associated with hemeralopia (avitaminosis), and light projection may prove deficient. The same may also happen in defects of the visual field; for instance, in homonymous hemianopia. Light projection may be wrong when cataract is associated with glaucoma or congenital choroidal coloboma in the same eye. Cataract in eyes which show hypotension should not be operated on before pressure has been normalized by local and general treatment. Otherwise such eyes perish through the flare-up of latent ciliary inflammation and endanger also the other eye by sympathetic ophthalmitis. Cataract in eyes with recent inflammation, however slight, should not be submitted to operation with the exception of cataract in heterochromia of the iris, in which there is no risk during and after operation.

N. BLATT.

POSTOPERATIVE COMPLICATIONS IN SENILE CATARACT AS CARRIED OUT IN THE OPHTHALMOLOGIC CLINIC OF BUCHAREST FROM 1920 TO 1928. FLORESCU and MANOLESCU, Cluj. med., 1929, no. 6.

The postoperative complications of senile cataract are partly of local (ocular) and partly of general nature. The ocular complications are noninfectious or infectious. Under the noninfectious complications are classified: the slow restoration of the anterior chamber (found in 4.87 per cent); opacities of the cornea due to folding of Descemet's membrane (in 11 per cent of all cases); hemorrhages due to cough, a blow in the eye or vomiting (in 24.18 per cent); rupture of the corneoscleral wound (in 0.97 per cent), vision being lost in 4 such cases; iris prolapse (in 4.38 per cent), entailing sympathetic ophthalmitis in 1 case; separation of the choroid in only 1 case; detachment of the retina in 1 case; intra-ocular hypertension (in 0.81 per cent) whereby all these cases terminated badly; spastic entropion (in 0.81 per cent); postoperative high degree astigmatism (in 5.25 per cent); secondary cataract (in 14.89 per cent). The authors class the following incidents among infectious postoperative complications: blepharoconjunctivitis (in 1.29 per cent), these cases being for the most part healed by oxycyanate irrigations, and suppurative keratitis in 8 of 616 operations, all of them ending by the loss of the eye, 1 of these by panophthalmitis; iridocyclitis (in 8.92 per cent), among which there were 8 suppurative cases and 1 case caused by sympathetic ophthalmia. Of the general postoperative complications the authors mention vomiting, cough, retention of urine and psychic troubles, all of which can have unfavorable effects on the ocular processes. N. BLATT.

BLOOD SUGAR ESTIMATION IN PATIENTS HAVING SENILE CORTICAL CATARACT. C. L. ANDERSEN, Acta ophth. **7**: 339, 1929.

Carefully compiled statistical records of the examination of 779 nondiabetic and 292 diabetic persons are arranged and plotted by Andersen. The results of this examination are compared with those presented by Gallus, Jacobsen and others. A review of the methods previously employed for blood sugar determinations and the procedure adopted for producing glycosuria and glycosemia are discussed. He gives his patient 50 Gm. of fresh white bread without butter and one-half glass of water as a test meal before the sugar determination is made. The Hagedorn-Jensen method is used for estimating the blood sugars and the Benedict and Fehling method for making determinations on the urine. He classifies the patients according to age. From the work of others and his own, he adopts an average sugar level for all the decades. The lenticular opacities are meticulously arranged according to their location and extensiveness in this review. He concludes that the frequency of cataract in persons with diabetes after the fortieth year is not perceptibly different from that in the nondiabetic person. In the decade from 51 to 60 there is a slight increase in the cataract in persons with diabetes, but in the later age periods this relationship is reversed. He could not establish any definite increase of carbohydrate metabolism in patients presenting senile cortical cataracts. He observed that the

sugar metabolism curve was approximately the same in persons having senile cataracts and in those in whom lenticular changes were not present.

A. M. YUDKIN.

Vitreous

THE SIGNIFICANCE OF VITREOUS OPACITIES. T. L. DE COURCY, Brit. M. J. 2:999 (Nov. 30) 1929.

According to the author, the embryologic studies of Miss Mann have taught us that the adult vitreous consists of a primary portion filling the retrolental space and passing along Cloquet's canal, a secondary vitreous filling in the space up to the internal limiting membrane of the retina, and a tertiary forming the suspensory ligament of the lens. The vitreous body is not enclosed by a structureless hyaloid membrane, being separated from the retina only by the internal limiting membrane. The slit-lamp furthermore shows some of the constituents of the vitreous, which appear as fibrils and nodules. As it disorganizes, the fibrils move more rapidly, and change their appearance to that of fibers of cotton-wool; the framework is altered, and pigmentation arises. The chemical and physical proportions have been studied especially by Duke-Elder, and show this body to be a colloid gel lying in the meshes of a framework, resembling the aqueous but with the addition of two proteins. Duke-Elder argues that some internal variation in the economy of the vitreous makes the gel fluid with the separation of its protein base in the form of strands, films or strings. These variations are probably due to a change of salt content, or a failure in the efficiency of an autoxidation system. This explains the largest number of vitreous opacities. Hemorrhages and exudates result from a lesion of the endothelial capillary walls.

The author suggests a classification of vitreous opacities as due to (1) changes in the capillary walls, (2) changes in the blood, (3) changes in the vitreous. Changes in the capillaries are exemplified by arteriosclerosis. Changes in the condition of the blood which in turn cause endothelial damage and leakage are observed in cyclitis due to focal infection, syphilis, excessive lactation, blood diseases with glandular enlargement and ductless gland involvements. Vitreous changes are congenital remnants, trauma without causing hemorrhage and mechanical obstruction, as in venous thrombosis producing hemorrhages.

To judge of the frequency of vitreous opacities, the author has taken 100 consecutive cases in which an ophthalmic examination was possible. They were present in thirty-seven. In a second 100, the percentage was the same. The method of examining for vitreous opacities with the ophthalmoscope is then described, and variations in the subjective symptoms are commented on. Their importance in myopia, especially from a prognostic point of view, and their association with changes in the lens are taken up.

The author believes that asteroid bodies are more frequent than the literature suggests, and are much more common than synchysis scintillans. It is not understood why some vitreous opacities are completely absorbed, while others remain easily recognizable. Absorption may be hastened by the administration of iodides internally, ethylmorphine

hydrochloride externally, aspiration and, of course, the removal of the infective source, if possible.

The author concludes that this subject has not had its adequate place in the literature and that with modern and more thorough methods of examination it is a field capable of much exploration.

A. KNAPP.

FORM AND STRUCTURE OF THE VITREOUS AS SEEN BY THE SLIT-LAMP. C. DEJEAN, Arch. d'opht. **46:** 477 (Aug.) 1929.

The author compares the structure of the vitreous to sheets of glass. He points out that a sheet of glass is transparent when looked at from the front but is opalescent when its edge is observed. The appearance of the vitreous as seen by the slit-lamp depends, then, on the intensity and angle of incidence of the light. He suggests that the confusion in terminology has arisen as a result. The vitreous as it appears in youth and old age is described. To support his conception further he has examined vitreous removed from an enucleated eye with the slit-lamp. The practical application of his investigation is to point out that the classic procedure of cutting off prolapsing vitreous allows the escape of fluid held within the sheets of transparent layers. He thinks that it is better to attempt to reduce the herniated vitreous. Following this the wound can be covered with conjunctiva. On several occasions he has noted that the inclusion of vitreous in the wound has not led to serious consequence.

S. B. MARLOW.

Uvea

TRAUMATIC PURULENT UVEITIS FOLLOWED BY GENERAL SEPTICOPYEMIC SYMPTOMS. S. DEGGELLER, Klin. Monatsbl. f. Augenh. **83:** 483 (Oct.-Nov.) 1929.

A large chip of iron, weighing 2 Gm., was removed by means of the magnet from the right eye of a man, aged 43, on the day following the accident. Shortly afterward the presence of purulent ophthalmitis became evident. Evisceration of the globe was contemplated but was refused by the patient. A gland above the right clavicle became painful and greatly enlarged three weeks after the accident. Glands on other parts of the body became affected, septic arthritis of the first toe of the right foot set in and atrophy of the globe was observed. The temperature and the heart remained normal. The blood showed staphylococci. The general symptoms disappeared within ten days. The second eye was not affected at the time of publication of the article.

K. L. STOLL.

EXCLUSION OF PUPIL. IRIS BOMBE. TRANSFIXION. RASVAN, Cluj. med., 1929, no. 6.

The author reports a case of fine deposits on the posterior surface of the cornea, with the iris discolored with washed out markings. With the loupe atrophic, white, bandlike vessels of the iris were visible. The pupil was round and immovable, and was covered with exudate. The vision equaled counting fingers at 0.75 meters. The tension was 32 mm. The Wassermann reaction was negative. The cutireaction

was positive. Operation was done by transfixion; vision was raised to counting fingers at 1.5 meters and tension was lowered to 10 mm. Thus in appropriate cases vision can be improved and a new artificial pupil created by transfixion.

N. BLATT.

Glaucoma

BUPHTHALMOS AND NAEVUS. T. R. AYNSLEY, Brit. J. Ophth. **13:** 612 (Dec.) 1929.

This is a report of four cases of unilateral buphthalmos occurring in mentally deficient children with hemiplegia. In three of the cases roentgenograms were made. In two there was a calcified meningeal nevus, and in one there was the suggestion of a meningeal nevus. The relation of nevi to glaucoma is discussed. The author concludes that facial nevus has in itself no causal relationship to glaucoma but that when the general cause of facial and bodily nevi, either a toxin or (as Unna suggests) slight trauma to the branchial clefts, acts to produce nevi of the cerebral vessels, then the mesoblastic structures of the developing eye may be affected; and this may lead either to imperfect formation of Schlemm's canal or the filtration angle, or to a coloboma of the disk and chorioid, or to a telangiectatic condition of parts of the intra-ocular vascular system, which may sooner or later, through growth, sclerosis, transudation or hemorrhage, lead to a disturbance of balance of the intra-ocular fluids and the production of increased tension.

The author urges, with others, that cases of nevi, particularly fifth nerve distribution, cases of buphthalmos with facial or bodily nevi, and cases of Hipple's disease should be examined by means of the x-rays and neurologically for the presence of intracranial lesions.

W. ZENTMAYER.

Retina; Optic Nerve

CAVERNOUS ATROPHY OF THE OPTIC NERVE AFTER EXCESSIVE STRETCHING. H. MITA, Arch. f. Ophth. **123:** 258 (Dec.) 1929.

The author carried out experiments on rabbits in which he studied the influence of luxation of the bulb in front of the palpebral fissure. In all the experiments the luxation was followed by a transient rise of the intra-ocular tension which went back to normal within twelve hours after the experiment had been started. In spite of a protective bandage over the luxated eyeball, the cornea was damaged within a few days. Special attention was paid to the histologic observations in the optic nerves of these rabbits, which were killed from three to eighteen days after the eyeball had been brought in the abnormal position. Anatomically, the optic nerve on the side of the luxated eye was found from 7 to 9 mm. longer than that on the other side. In all of the rabbits treated in the manner described and examined histologically cavernous atrophy of the optic nerve was found. The caverns were small and found mostly in the prelaminar or the intralaminar part of the optic nerve. The caverns usually appeared only in one sector of the optic nerve. This sector always corresponded to the part of the disk where the scleral border was sharpest and formed a real spur. The author concludes that cavernous atrophy is a symptom of excessive stretching

of the optic nerve which may be due to trauma or dislocation of the bulb (orbital tumors). His observations speak for Stock's theory that glaucomatous atrophy is caused by a stretching of the single fibers because of the receding lamina cribrosa. The fibers must tear off, because it is impossible for them to slip through the holes in the lamina, which are much too small for the thick medullated retrolaminar fibers.

P. KRONFELD.

A CASE OF TAPETORETINAL DEGENERATION WITH CONELIKE FORMATION AT THE MACULA. H. RIEGER, Ztschr. f. Augenh. **70**: 22 (Dec.) 1929.

Rieger describes a conelike formation which he observed at the macula in the eye of a young woman. A similar picture of the fundus was reported by Metzger, who attributed the retinal destruction to a cystic degeneration probably induced by an injury to the frontal bone above the eye. Rieger does not classify this case as one of injury, but as one of the heredomacular degeneration type and similar to his. The change is probably due to a hyperplasia of the glial tissue about the fovea. He attributes the fine granular-like bodies which were noted in the periphery of the retina to some form of a tapetoretinal degeneration rather than to a syphilitic lesion of the tissue, even though his patient gave a positive Wassermann reaction of the blood. His case differs considerably from the syphilitic changes in the retina that are described in the literature. He concludes that his case cannot be classified as a syphilitic disturbance, but should be considered as an hereditary degenerative process of the retina.

A. M. YUDKIN.

Neurology

COPPER AND SILVER STORED IN THE EYE, LIVER, SPLEEN AND KIDNEYS IN PSEUDOSCLEROSIS. A. VOGT, Klin. Monatsbl. f. Augenh. **83**: 417 (Oct.-Nov.) 1929.

Referring to his article on the pigment ring in Descemet's membrane (abstracted in ARCH. OPHTH. **2**: 480 [Oct.] 1929), the author reports the postmortem examination of one patient who had been under observation for more than forty years. This woman died recently at the age of 61. Chemical examination yielded 29 mg. of copper and 1.2 mg. of silver per hundred grams of liver, against 10 mg. of silver and only 1.7 mg. of copper per hundred grams of kidney. The spleen contained 1.4 mg. of copper and 2.5 mg. of silver. These observations prove that in pseudosclerosis metals consumed in the food cannot be eliminated. The author puts especial stress on the fact that this research work was possible only by the help of the slit-lamp microscope. The globes, placed in formol forty minutes post mortem, were embedded in paraffin. The substratum of the pigment ring consisted of silver, evidenced by cyanide of potassium. The "copper cataract" was actually caused by copper. Vogt recently found silver in Descemet's membrane in a case in which internal doses of this metal were given, and in two cases of argyrosis of the conjunctiva. Illustrations of these three cases may be found in the second edition of Vogt's "Atlas of Slitlamp Microscopy."

K. L. STOLL.

TUMORS IN THE REGION OF THE PITUITARY. K. MYLIUS, Ztschr. f. Augenh. **70**: 9 (Dec.) 1929.

A comprehensive study was made of a case of a patient, aged 19. Polydipsia, polyuria, progressive bilateral optic atrophy, marked lethargy and increasing cachexia predominated until death, when increased prostration and disturbance of temperature were also observed. Definite remissions of the symptoms were noted from time to time. The cachexia was observed from the beginning of the illness, which lasted for a year. Mylius does not believe that the cachexia was due to disturbance of secretion produced in the frontal portion of the pituitary glands, but to a disturbance in infundibular region and probably a cerebellar lesion. Previously recorded cases show that the symptom complex of polyuria, polydipsia and lethargy may be produced by a pathologic disturbance in this area. The neurologic literature also reveals the fact that there may be a regulating center for sleep and wakefulness in the gray matter in the floor of the third ventricle. The administration of pituitary extract does not alleviate the foregoing manifestations when they are due to a lesion at the base of the brain. The clinical diagnosis was confirmed at necropsy by the finding of a cystic tumor involving the midbrain with pressure on the optic tract; the third ventricle and basal ganglion were found, and the pituitary body was intact. The whole picture was that of a disturbance in the region of the midbrain rather than a pituitary disturbance.

A. M. YUDKIN

Tumors

HEMANGIOMA OF THE ORBIT. D. SHAPIRO, Am. J. Ophth. **12**: 901 (Nov.) 1929.

A girl, aged 12, had had proptosis of the right eye since birth and this had gradually increased. The eye was displaced downward, inward and forward. The lower lid covered the lower, inner third of the pupil. The eye could be closed by effort. There was an eversion of soft but not fluctuating tissue in the upper fornix, and the lacrimal gland protruded from the orbit. Vision was 20/60. Ophthalmoscopy gave negative results except for slight engorgement of the retinal veins. The left eye was normal.

A horizontal semicircular incision was made in the conjunctiva, extending from the inner canthus upward into the fornix and down to the level of the outer canthus. The conjunctiva was dissected out en masse. Six weeks after operation the eye could be opened very well, the levator had almost completely recovered, and the external rectus had improved so that the globe could be carried out beyond the midline. Vision was unchanged. The pathologic diagnosis was: benign hemangioma (partly hyalinized and fibrotic).

W. S. REESE.

TRAUMA IN THE GENESIS OF ORBITAL SARCOMA. E. MORETTI, Ann. di ottal. e clin. ocul. **57**: 515 (June-July) 1929.

A girl, aged 12, was struck in the eye by a potato. Ten days later a swelling of the upper lid and inner part of the orbit developed, and during two months this increased to the size of a lemon, involving both lids and covering the globe completely. Fever and swelling of the

glands suggested an inflammatory process, but puncture allowed the escape of only a small amount of pus and tissue composed chiefly of small round cells, allowing the diagnosis of sarcoma. An attempt to eviscerate the orbit was abandoned because of excessive hemorrhage. Death occurred one month later; radium had no effect. A statistical study reveals contradictory opinions as to the etiologic importance of trauma in malignant tumors. The author believes that it has not been proved that a single traumatism ever produced a malignant growth, but that it has, in some cases, caused an acceleration in the growth of a previously slow-growing tumor, as doubtless occurred in the present case. (Bibliography.)

S. R. GIFFORD.

Injuries

PERFORATING INJURIES OF THE EYE BY SMALL STEEL FRAGMENTS. H. L. BEGLE, Am. J. Ophth. **12:** 970 (Dec.) 1929.

The author, who has evidently had a very extensive experience in this work, divides the subject into the following: (1) the damage done to the eye in foreign body injuries is directly proportional to the size of the foreign body; (2) injuries by small foreign bodies perforating the cornea near the limbus; (3) removal of small steel fragments from the lens without cataract formation; (4) difficulty in removing steel fragments from the lens after the capsular wound is healed; (5) possibility of a small foreign body of steel undergoing complete rusting in the eyeball, and through resorption of the rust eventually disappearing; (6) minimal damage by small foreign bodies completely traversing the lens.

W. S. REESE.

NOTE ON TRAUMATIC ASPHYXIA WITH OCULAR COMPLICATIONS. J. B. HAMILTON, Brit. J. Ophth. **14:** 22 (Jan.) 1930.

A boy, aged 15, was caught between the door and the floor of a lift, and his chest was severely compressed for a period of not more than sixty seconds. Three hours later, his face was swollen, and his eyes were bloodshot. These symptoms increased in the next twenty-four hours, accompanied by drowsiness and loss of memory. Vision seemed unaffected. When the patient was seen by the author eleven days after the accident, there was symmetrical bilateral conjunctival hemorrhage but no vascular changes in the fundi. Vision was normal in each eye.

The outstanding feature of the case as compared with similar cases reported is the surprisingly short duration of the compression of the chest which resulted in such extensive latent signs.

W. ZENTMAYER.

THE DISAPPEARANCE OF COPPER DEPOSITS FROM THE EYE. A. JESS, Ztschr. f. Augenh. **69:** 59 (Sept.) 1929.

Following an injury to the eye by copper or its alloy, brass, the ocular tissue becomes impregnated by copper salts. Jess observes that these deposits in the cells eventually disappear from the tissue. This is not the case, however, with iron deposits. The rapidity of disappearance depends on the amount of metal in the tissue and the age of the person. Youthful eyes seem to repair much faster than adult eyes.

These observations are substantiated by observations recorded in many similar cases, clinical and experimental. He notes that an eye often retains useful vision if no irritative iridocyclitis or glaucoma sets in, and that sympathetic ophthalmia does not, as a rule, accompany injuries produced by copper deposits. Jess concludes that it would be safe to leave a small piece of copper or brass splinter in the eye, providing it does not set up an irritative process or increase intra-ocular tension.

A. M. YUDKIN.

Therapeutics

SOME INDICATIONS FOR TARSORRHAPHY. VAN LINT, Arch. d'opht. **46:** 411 (July) 1929.

This procedure has been commonly used in neuroparalytic keratitis and lagophthalmos. The author recounts his experiences in cases of chronic and serpiginous ulcer, traumatic staphyloma of the sclera and contused perforating wounds of the anterior segment. He has also used this method following cataract extraction, and has been astonished by the freedom from reaction when the bandages were removed. As the result of its use in several cases of torpid ulcer of the cornea, he has noted the subsidence of the subjective symptoms: pain, lacrimation and photophobia. He feels that simple closure of the lids is the only treatment necessary, and that ointments and collyria, mydriatic and antiseptic, are superfluous. He has had the best results in the conditions mentioned, and feels that the procedure should be used more frequently than it is.

S. B. MARLOW.

CALCIUM THERAPY IN PHLYCTENULAR DISEASES. M. CREMER, Arch. f. Augenh. **100-101:** 729, 1929.

The author treated a series of seventy-five patients with phlyctenular keratoconjunctivitis by the usual clinical methods and a second series of seventy-five patients by the same method with the addition of calcium by mouth. He found the most palatable calcium preparation to be a proprietary medicine called Calcium-Sandoz. This preparation contains about 9.3 per cent of calcium bound to gluconic acid.

The rationale of calcium therapy is based on the fact that this drug alters the permeability of the smaller blood vessels, and perhaps the lymph vessels. It has been found of use in the prevention of eczema. He points out the difficulties of judging accurately the success of any form of therapy in phlyctenular disease and takes as his criterion the length of stay of the patient in the hospital. The average stay for patients not given the calcium was 22.5 days; that for those given calcium was 14.1 days.

F. H. ADLER.

ULCUS SERPENS TREATED WITH OIL OF WINTERGREEN. K. SABATZKY, Klin. Monatsbl. f. Augenh. **83:** 498 (Oct.-Nov.) 1929.

The author reported his previous results in the treatment for serpent ulcers of the cornea with synthetic oil of wintergreen in the *Klinische Monatsblätter für Augenheilkunde* (vol. 81, p. 274) and before the International Congress at Amsterdam. Recently he obtained encouraging results in which the cornea remained transparent and recovery

occurred promptly. In only one of twenty-four cases of pneumococcus infection, puncture of the anterior chamber had to be performed. In all cases but the one the vision was 5/6, while the cornea showed only slight opacities. Facettes and astigmatism were not observed.

The oil is applied after the ulcer has been cleansed and scraped under cocaine anesthesia. This process is followed by a massage with a spatula, the oil being forced into the corneal tissue. Atropine ointment and a bandage complete the treatment. One application was usually sufficient. The author thinks that oil of wintergreen, besides being a germicide, induces a formation of new and clear corneal tissue, and that it lowers the tension of the globe, even during the copious instillation of atropine. The oil and the protoplasm of the cells, in his opinion, form a close chemical combination in the newly formed corneal lamellae and so prevent the formation of opaque tissues. K. L. STOLL.

IONTOPHORESIS IN CATARACTS. B. V. PROTOPOPOFF, Arch. oft. **6**: 378 (Oct.) 1929.

The author treated cataract in twenty-eight eyes of eleven women and four men by Wirtz's iontophoresis method (the negative electrode dipped in a 1 per cent solution of sodium iodide in contact with the cornea through a thin cotton layer, and the inactive pole held by the patient at the opposite temple). In twenty-two eyes there was incipient cataract, in two, mature cataract, and in four, complicated cataract.

Each patient was examined with the slit-lamp before and after the treatment, and also several months after the treatment had been finished.

None of the cases showed diminution of the opacity of the lens. The two non-mature cataracts had to be operated on within eight to ten months. The four cases of complicated cataract continued to progress. All of the cases of incipient cataract remained stationary for one year, the period of observation.

The author's conclusion is that iontophoresis may delay the development of incipient cataracts only. But further study and observation of a larger number of patients are required. O. SITCHEVSKA.

Blind

CAUSES AND PREVENTION OF BLINDNESS IN COLORADO. E. JACKSON, Am. J. Ophth. **12**: 965 (Dec.) 1929.

The author pleads for accurate statistics so that appropriate measures for preventing blindness may be instituted, and he discusses the difficulties in obtaining such statistics. He concludes that the prevention of blindness depends on (1) persuading patients to seek good treatment at the earliest possible time; (2) the readiness of physicians to recognize disease that may lead to blindness, when the eyes seem to be little if at all affected; (3) the understanding that diseases which do not usually affect the eye may in some cases cause blindness, and need to be watched with this fact in mind; (4) the preparation of eye physicians to recognize the possible dangers of the conditions they treat; and their willingness to take responsibility for deciding when an eye should be

taken out because it may endanger the sight of its fellow; or to operate, as for cataract or glaucoma, when this alone will prevent blindness.

W. S. REESE.

PROPOSED DEFINITION OF BLINDNESS. J. A. M. A. **94:** 568 (Feb. 22) 1930.

Adoption of a general standard of blindness throughout the world is recommended in a report from the Health Organization of the League of Nations. The recommendation is included in a study of the welfare of the blind in twenty-six countries, and the object is to secure greater uniformity in the treatment of the blind for the comparison of statistics of blindness between various countries, and to show more precisely the extent of the real administrative problem of the blind in various countries. For the purpose of the United States census, instructions to enumerators of 1920 were as follows: "Include as blind any persons who cannot see well enough to read even with the aid of glasses. The test in the case of infants must be whether they can apparently distinguish forms and objects, and, in the case of older persons who are illiterate, whether they can presumably see well enough to read if they knew how to read. Do not include any person who is blind in one eye only." A conservative figure of 2,390,000 is quoted as a "probable underestimate" made in 1910. In those countries in which a census of the blind cannot be obtained, the prevalence of blindness is frequently greater. The practicability of the adoption of a uniform definition is questioned also in the report, because a change of definition in countries in which public assistance is given to the blind as such would have one of two effects: It would either deprive some persons of the benefits which they are now receiving or it would involve the public in increased expenditure. But if the attempt were made, the first step would be to decide how many definitions are needed and for what purpose, as for census purposes, educational purposes and the purpose of adults in such matters as training and employment and financial assistance.

W. ZENTMAYER.

General

PSEUDOFOREIGN BODIES OF THE EYE. H. COPPEZ, Arch. d'opht. **46:** 449 (Aug.) 1929.

The author points out that this subject has become of increasing importance since the establishment of the compensation laws. Very little has been written about it. He divides the cases into three groups: (1) subjective, (2) objective and (3) pseudoforeign bodies with a foreign body in another location. In the first group are cases of beginning interstitial keratitis and acute iritis. In these the length of time elapsed between the date of the injury and the reporting of the accident permits the determination as to whether or not trauma actually occurred. In the second group, pigment deposits, old synechiae and atrophy of the iris have been mistaken for foreign bodies. He points out that great care is necessary in the examination of these cases. The third group deals with intra-ocular changes revealed by the ophthalmoscope with misleading roentgenologic observations. An interesting group of cases illustrating the points made is included.

S. B. MARLOW.

THE SIGNIFICANCE OF THE BLOOD CORPUSCLE SEDIMENTATION TEST IN OCULAR DISEASE. HANS SCHMELZER, Ztschr. f. Augenh. **70:** 149 (Jan.) 1930.

Twenty-five different ocular diseases were investigated by Schmelzer in order to determine the possible assistance this procedure might lend to ocular diagnosis and prognosis. He noted that in cases in which increased sedimentation was present, the ocular disease was due to some constitutional disturbance and in those in which the etiology was definitely determined as local, some concurrent internal malady was present, such as tuberculosis, emphysema, bronchitis or chronic nephritis. Sedimentation was normal in persons with senile lenticular changes and in glaucoma. In dacryocystitis, abscess of the lid, ulcus serpens and herpes of the cornea strongly positive reactions were given to the sedimentation tests. After treatment, the positive observations usually became normal. The procedure has no significance in differentiating between pseudoglioma and glioma. This method, as far as can be determined, has no real differential diagnostic value in ophthalmology, but it may be used to ascertain whether the condition is getting better or worse.

A. M. YUDKIN.

Hygiene, Sociology, Education and History

INDEMNITY FOR LOSS OF THE SECOND EYE AFTER PREVIOUS LOSS OF THE FIRST EYE. L. MAGGIORE, Ann. di ottal. e clin. ocul. **57:** 557 (June-July) 1929.

On the basis of a case, the author discusses legal decisions in similar cases. His conclusions, which were borne out by the court, are as follows: When a worker has suffered a reduction in wages after the loss of his first eye, damages on the basis of 100 per cent of the lost salary should be awarded for loss of the second eye. When no reduction of wages has been suffered, indemnity should be on the basis of 65 per cent of the last salary, since he has already been indemnified on the basis of 35 per cent for the loss of his first eye, without having a corresponding loss of earning power. If he were paid on the basis of 100 per cent, the total damages would amount to 135 per cent, which is not allowed by law.

S. R. GIFFORD.

Society Transactions

EDITED BY DR. GEORGE S. DERBY

COLLEGE OF PHYSICIANS OF PHILADELPHIA
SECTION ON OPHTHALMOLOGY

A. G. FEWELL, M.D., *Reporter*

Jan. 16, 1930

H. MAXWELL LANGDON, M.D., *Chairman, Presiding*

IRIDECTOMY IN BULLOUS KERATITIS. DR. WILLIAM ZENTMAYER.

A man, aged 68, had relapsing bullous keratitis. The condition was of about one year's duration when an iridectomy was done which had apparently resulted in a cure, as no recurrences had taken place within eight months, and the eye was functioning normally with a visual acuity of 6/15.

The patient had been constant and faithful in attention to the eye, having been regularly in attendance at one clinic for six months, where the usual procedures, including irradiation and the thermophore, were used, without benefit. The etiology was obscure. The only possible contributing cause was an ethmoiditis for which an operation has been done successfully early in the disturbance of the eye. Aside from the relapsing bullae with the accompanying severe subjective symptoms, temporary slight increase in tension and an alteration in the anterior lens capsule, the eye was normal.

DISCUSSION

DR. COWAN: I saw this man quite a while before Dr. Zentmayer at the Wills Hospital, and was very much interested because he presented the same condition as that which Dr. Holloway and I showed some time before at the section. There was a bluish-white, punctate deposit on the anterior surface of the lens, forming a sort of lacework design. The ragged edges extended forward into the anterior chamber. At present there is a difference of opinion as to the etiology of these cases.

The same type of case is illustrated by Bedell in the Jackson Anniversary Book; he called it a proliferation of the superficial layers of the lens capsule. Vogt, in his atlas, illustrates the same condition and considers it a peculiar form of retained pupillary membrane. He pointed out that the design roughly outlines the arrangement of vessels of the tunica vasculosa lentis. Dr. Holloway and I, agreeing with Vogt, presented our case as one of persistent pupillary membrane. Since then Vogt has reported several other cases of the same kind, but he now considers the exudate due to a degeneration of the superficial lens capsule. Meesman, in his atlas, illustrates the same condition and believes as does

Vogt. Kraupa includes these cases with glassblower's cataract, and believes that the exudate is due to a glandular degeneration of the capsule lamella. Schiek believes that the exudate is a deposit of cells from the posterior surface of the iris.

The first case that I saw was one of Dr. Ring's. Shortly after this I saw a case at the University, and then the one presented, all within a very short time. I think that these cases are much more common than is supposed. They always occur in elderly people, with other degenerative changes and often with glaucoma. The patient presented by Dr. Holloway and myself developed glaucoma a few days after he was shown.

DR. GRISCOM: Some years ago, I reported a case of bullous keratitis before the Pennsylvania State Medical Society, in which the history was similar to that of Dr. Zentmayer's patient. The patient was a woman, aged about 60, who came to the Wills Hospital with recurrent bullae of the cornea, which repeatedly ruptured and reformed, accompanied by intense pain. There was a rise of tension in this case, but no other evidence of ocular disease aside from the bullae.

All efforts to relieve the patient, including iridectomy, were of no avail, and it was finally decided that the eye should be enucleated because of the intense pain. In the course of the examinations prior to this operation, it was discovered that she had an active ethmoiditis. She was treated for this condition, and whether or not because of this treatment, the bullae ceased to form and the eye remained quiet.

I refer to the foregoing history because after one and a half years the patient returned to the hospital with a similar condition in the other eye. In spite of all treatment, including treatment of the ethmoids which were not actively diseased at that time, she continued to have repeated attacks. The patient finally became discouraged and disappeared, but it is likely that some one has since enucleated the eye.

DR. DESCHWEINITZ: Dr. Zentmayer has achieved an admirable operative result. A young Jew who was long the subject of relapsing bullous keratitis which had resisted all the usual nonoperative procedures was finally cured when after an unusually large bulla had formed all the loosened epithelial layer was removed, and the exposed surface was painted with a 1:20 solution of trichloracetic acid, applied especially along its edges.

No definite cause for the formation of these large bullae was discovered. The patient was highly myopic and dwelt in a malarial region, and had frequently received full doses of quinine, but prior to the cauterization described these had had no salutory influence on the keratitis. The slight corneal haze which remained caused no noteworthy visual inconvenience.

DR. WILLIAM ZENTMAYER: Dr. Buchanan has brought up the question of malaria as a cause of the bullous keratitis. The patient said that many years ago he had a "touch of malaria," but this seemed too long ago to have any bearing on the etiology of the corneal condition.

A CASE OF ELECTRIC CATARACT. DR. HOLLOWAY.

Dr Holloway presented the history of a case of electric cataract, the essential points of which were as follows: A boy was subjected to a

shock from an electric wire carrying 2,200 volts. As far as could be ascertained, inflammatory signs had never been present. Failure of vision was first noted nineteen months after the injury, and when the patient was first seen by Dr. Holloway ten months later, or twenty-nine months after the accident, there was complete opacification of each lens. There was a small central capsular plaque on each cataract.

Following iridectomy and evacuation of the lens, the patient had 6/12 vision with the right eye and 6/6 with the left. No demonstrable lesions of the fundus were present in either eye.

Dr. Holloway said that he intentionally avoided saying anything about the pathogenesis of this type of cataract, as he hoped that some of the men interested in biochemistry and pathology would discuss this point. As far as he knew, it was probable that the observations of Hess were those generally accepted. This observer had found that subsequent to an electric discharge, there occurred a distortion of the capsular epithelium with the accumulation of fluid between the capsule and the lens fibers.

ADENOCARCINOMA OF CONJUNCTIVA. DR. EDWARD B. SPAETH.

Dr. Spaeth reported a case in which the growth was of at least one year's duration, and in the past few weeks it was showing a very rapid increase in size. The greatest part of its size at the time of presentation had been reached in the past three weeks. At the first examination the tumor mass extended from the limbus, where it had grown over the cornea for about 2 mm., toward the outer canthus for a bit more than three fourths of the scleral surface, and in width it was slightly more than that of the normal palpebral fissure. It was elevated at its highest point for several millimeters, multilobed, soft and with a very copious blood supply. It was firmly adherent to the underlying sclera.

The limbus is the most common site for epibulbar carcinomas. In this case there is, therefore, nothing unusual as to position. Also epibulbar carcinomas, as a rule, respond rather readily to treatment with radium. They are rather rapid in their growth. Adherence to the underlying tissues occurs late in their course, and they usually extend completely about the limbus before penetration occurs because of the resistant sclera. In this case, there were a few points of difference from these usual features. These were: very slow growth, penetration to the underlying tissues, very copious blood supply, the brown granules of pigment scattered throughout the tumor mass and unusual resistence to radium therapy. Radium was applied by conjunctival contact three times at two week intervals with but little change in size or appearance. Ten days after the third application, the growth was resected from the sclera by actual cautery. A fourth application of radium was made the same day. The case then went on to uneventful recovery and the patient has remained well for six months.

Judging from the duration of the carcinoma and its rapid growth, it is possible that the tumor started originally with an epithelial plaque, a benign adenoma starting from glands of a rudimentary acinotubular character or from a nonpigmented mole. The late sudden growth would indicate the time at which malignancy developed therein. This

is also consistent with the pathologic report made on the sections. An incipient cataract remained unchanged throughout the course of the treatment. Vision was still 6/5 with correction.

A Method of Uncovering Latent Hyperphoria. Dr. Leighton F. Appleman.

For uncovering latent hyperphoria Dr. Appleman presented a method which consists essentially of three steps: 1. With a Maddox rod the muscle balance is tested at a distance of 6 meters. This test reveals the manifest error at this distance. If a deviation from the normal is present, a prism is placed before the eye in such a manner as to obtain equilibrium. 2. The patient is given a card on which is the dot-and-line of Graefe. He is told to hold this at the reading point with the line horizontal in testing for hyperphoria. A 20 degree prism is then held in front of one eye with its base toward the nose. Lateral diplopia is thus produced, and, if no hyperphoria is present, the images will be on the same horizontal plane. Should one be higher, a prism of sufficient strength is placed before one eye to bring the images on the same horizontal plane. 3. Leaving this prism on, the patient is told to look at the point of light 6 meters distant or across the room. The Maddox rod is again used, and if the patient now shows an overcorrection, the strength of the prism is reduced by half degrees until the eyes are again in equilibrium. This constitutes the second distance estimation. The difference between this and the amount shown by the first test constitutes the amount of latent deviation.

In an analysis of 500 cases, there were 253 in which primarily no hyperphoria was shown, but in which from 0.75 to 3 degrees were uncovered by means of this method of testing. In the remainder, while a low degree of deviation was found at the first test, considerably more was revealed as latent. Incorporation of these observations in the lenses for constant wear produced gratifying relief from symptoms for which the patients sought relief.

DISCUSSION

Dr. William Zentmayer: The objection that came to my mind was to the high prism used. The slightest angulation of the prism may produce this apparently latent hyperphoria, but I suppose that care was taken to see that this did not occur.

Although I do not wish to put my opinion above that of Mr. Maddox, supported by Dr. Appleman, that the correction of hyperphoria reduces the amount of lateral deviation, in my experience it has no such effect. In my opinion, the correction of hyperphoria under 2 degrees is rarely necessary. I consider than when refraction work is done thoroughly, it is seldom necessary to give special attention to the correction of a low degree hyperphoria.

Dr. deSchweinitz: I am convinced that the prismatic correction of hyperphoria represents a highly important therapeutic procedure, often, moreover, when the hyperphoria is of small degree and the most accurate correction of the refractive error has failed to achieve the desired result.

In my experience and that of my associate, Dr. Fewell, when the hyperphoria is latent, the Marlow method of uncovering is a most satisfactory one. Having had no experience with Dr. Appleman's technic, I am unable to discuss it in relation to that advised by Marlow.

The late Dr. E. O. Shakespeare corrected hyperphorias with prisms at a period long antedating the well known studies of the late Dr. George Stevens. I am not sure, however, whether Dr. Shakespeare has placed his cases on record.

DR. HOLLOWAY: I have used Dr. Appleman's routine in several instances, and have found that my results are similar to those obtained by Dr. Appleman. As far as a prism is concerned, it is not necessary to use as high a prism as has been suggested. I believe that the test is essentially a fogging of the muscle, in the same sense that this word is used in a case of refraction. I think that all clinicians obtain satisfactory results from the use of vertical prisms, but occasionally one obtains just as satisfactory a result from taking off a prism that has previously been prescribed.

I consider the Marlow test a distinct acquisition to the methods of treatment for muscle imbalance, and I generally use it for several days. One of the most grateful patients I have had was one who had been wearing a vertical prism placed in the wrong direction, the error being uncovered by the Marlow test.

DR. FEWELL: In regard to the Marlow test, patients do object to wearing a bandage over an eye, but in patients who are already wearing glasses, by making one glass frosted, I have seen 0.25 degree of hyperphoria change to 1 or 2 degrees after the patient has worn this glass for one or two days. Patients will not object as much to this frosted glass as they would to the bandage.

DR. H. MAXWELL LANGDON: In the case which Dr. deSchweinitz referred to as having seen in consultation with me, a frosted glass was tried for a week, and for the next week a tight bandage over the eye was worn constantly during the waking hours. The patient found the bandage much more comfortable and less annoying than the frosted glass, and incidentally a hyperphoria of 3.5 degrees was developed, where less than one was noted previously.

DR. CARPENTER: I have the privilege of looking back over a good many years of ophthalmic practice and am rather astonished to find that agreement has not been reached as to the prism for vertical muscle deviation. I believe that every eye properly examined which shows a relatively high defect should be corrected by vertical prisms even after a most perfect correction of the refractive error.

There are some people who have such a splendid nervous system that they can go with hyperphoria or hyperopic astigmatism with glasses of 15 degrees or more of faulty placement of the cylinder and come back and sing the physician's praises. I met one patient who had 5 degrees left hyperphoria. I ordered prisms 2.5 degrees base up for the right eye and 2.5 degrees base down for the left. The patient was a very busy lawyer and did not return for two weeks, when I found that the prism 2.5 degrees base down was placed base up, so that he was wearing 5 degrees in the wrong direction and was delighted with the result.

I believe that hyperphoria is most important and should be treated and I do not see why the Maddox test cannot be used for the 13 inch test exactly as in the 6 meter test.

DR. LEIGHTON F. APPLEMAN: I think that the reason there is not more unity of opinion as to the value of prescribing vertical prisms is that the majority of examiners have not a definite method for uncovering these errors. While there are many different methods of estimating the muscle balance, and while it is probably true that some will get results by using one method or another, I believe that every one should stick to some one method by which these latent defects may be uncovered and corrected as part of each examination.

I was led to a recognition of the importance of correcting low degrees of hyperphoria in the course of the examination of many medical students who at a first examination took a certain correction and wore it comfortably for a year; at that time they began again to have discomfort and on examination under exactly similar conditions, showed exactly similar refractive errors as found the year previously excepting that a low degree of hyperphoria was uncovered. The prescribing of a vertical prism as low as 0.5 degree has enabled these patients to go on with the second year's work with perfect comfort.

In regard to the lateral deviations, it has been my experience that, aside from the need for convergence training by means of prism exercises, the lateral deviation usually takes care of itself.

In regard to the strength of prism used to obtain lateral diplopia in testing at the near point, I use a prism of 20 degrees. It is not necessary to use one this strong provided diplopia is produced, but it is with this one that I have been accustomed to work.

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

WILLIAM F. C. STEINBUGLER, M.D., *Secretary*

Jan. 20, 1930

ERNEST F. KING, M.D., *Chairman*

UNUSUAL OBSERVATIONS ON THE MUSCLE AFTER OPERATION ON THE FRONTAL SINUS. DR. JAMES WATSON WHITE.

Following operation for mucocele of the right frontal sinus at the Herman Knapp Memorial Eye Hospital, there developed a spasm of the right inferior oblique. At no time was there a paresis of the superior oblique. The head was tilted to the left shoulder and back with the face turned moderately to the right.

On examination, there was right hyperphoria of 25 degrees in the primary position; this increased in looking up and left, but was less in all other fields.

Three months after operation on the sinus, the right inferior oblique muscle was tenotomized. This corrected the vertical deviation almost entirely, but after one month, it was found to be increased. From then on, it gradually lessened until the patient was seen six months later, when there was no deviation except in extreme rotation up and left. For three years this remained the same. The spasm of the inferior oblique as the primary lesion is difficult to explain, there being no paralysis of the superior oblique muscle. Primary paresis of the left superior rectus is more probable, fixation being performed by the right eye. The prolonged occlusion of the right eye following the operation caused fixation with the left or paretic eye and secondary deviation of the right inferior oblique. The remaining right hyperphoria in looking up and to the left would bear out this opinion.

DISCUSSION

DR. ARNOLD KNAPP: I think that the one interesting problem that this case presents is the question of spasm. How soon can a spasm develop? We have a number of physiologists here; perhaps they may be able to tell us. In looking over a monograph of Bielschowsky, I found that he had observed paralysis of the inferior oblique after an operation on the frontal sinus, and that he regarded it as due to an interference with a connecting band which directly connected the trochlea with the inferior oblique. If this band exists, and if in certain patients it is interfered with sufficiently to cause a paralysis, why could it not under less marked conditions cause a spasm?

DR. CLYDE E. MCDANNALD: May I ask how long after the frontal sinus operation this tenotomy was done?

DR. JAMES WATSON WHITE: Four months.

In answer to Dr. Knapp's question as to how soon spasm develops, I can recall few primary spasms. In one case that I recall distinctly, a definite primary spasm occurred in a boy who was struck over the brow with a bale hook. The wound healed, leaving a thick scar. During the process of healing, the patient had a superior rectus spasm of this side that entirely cleared up.

BILATERAL SPASM OF SUPERIOR RECTUS MUSCLE. DR. FRANCIS W. SHINE.

A man, aged 42, first appeared at the New York Eye and Ear Infirmary on Dec. 9, 1929, giving a history of repeated attacks of this peculiar twitching during the previous three years. The lids of both eyes twitched and then closed so tightly that the patient could not open them again for a few minutes. Since then he had had at least one and sometimes as many as fifteen or twenty attacks each day. Sneezing, coughing, nervousness and excitement were usually sufficient to initiate an attack. Between the attacks the lids often twitch slightly. Diplopia or subjective visual disturbance had not occurred. The Wassermann reaction was negative.

On closure of the lids a spastic condition of the orbicularis and the superior recti of both eyes was seen, the eyeballs being elevated at times, far upward under the lids, where they remained for some time after

relaxation of the orbicularis. Between attacks the motility of the eyes was normal, as was the muscular balance. There was a manifest hyperopia of 1 diopter in each eye, with normal vision when corrected. The fields and the fundi were normal.

Neurologic examination by Dr. Poe pointed to ocular myotonia spastica. No similar condition was elicited elsewhere in the body. The reflexes were normal and the results of examination otherwise negative.

Examination of the nose and throat showed chronic tonsillitis and chronic ethmoiditis. There was considerable postnasal discharge, and a shadow in transillumination of the right antrum. This was washed out, yielding a small amount of pus.

DISCUSSION

DR. JAMES WATSON WHITE: Might not this case be one of spasm of associated movements? This is not infrequently seen as a forerunner of paralysis of associated movements.

DR. MARK J. SCHOENBERG: It does not seem to be a spasm of associated movements, for the reason that physiologically the function of the orbicularis is not associated with that of the superior rectus. On the other hand, when the patient gets one of these spasmodic conditions, he closes his eyes tight and then gradually opens his lids, and you see that his eyes are directed upward. At the same time the upper lids droop somewhat, while the lower lids are in normal position. So there is a spasm of the superior recti and perhaps an inhibition of the levatores palpebrae, an unusual condition.

I understood Dr. Shine to say that he does not think it is a nervous condition. It must be a nervous condition due to some inflammatory or toxic process going on in the ethmoids. The question of myotonia should also be considered; the only trouble is that myotonia is usually familial. Has an inquiry been made as to the existence of myotonia in his family?

DR. ARNOLD KNAPP: Is not the levator action a slight one, and is it not much less than that of the orbicularis? If a patient with facial paralysis is asked to close the eye, the cornea will move up without any levator action.

DR. SCHOENBERG: I do not see how the orbicularis alone can account for that. The lower lid does not seem to be displaced upward as it would be in a spasm of the orbicularis. It is only the upper lid that is lowered, and it is difficult to accept a partial spasm of the orbicularis involving only the upper half.

DR. FRANCIS W. SHINE: I think Dr. Schoenberg misunderstood me. I said I thought it was a nervous condition. It is probably hysterical. That is what I had regarded it as being, and it might well be initiated by such a condition as the patient had in his nose.

What the patient showed on presentation was not a true picture. Probably the superior recti had become somewhat weakened. The only cases anything like this that I have seen have followed epidemic encephalitis; one was reported recently by Warren Reese of Philadelphia. Dr. Hughes tried to paralyze the orbicularis in one eye with the injection

of alcohol, and he was successful in a number of cases, but in this case I cannot say that it has worked at all. I do not see any improvement. What I should like to know is whether we are justified in pursuing treatment on the nose in this condition.

A Case of Optic Atrophy in Paget's Disease (with Lantern Slides). Dr. Rudolf Aebli.

This paper will be published in full.

DISCUSSION

Dr. Ernest F. Krug: The literature reveals few case reports by oculists of eye complications in Paget's disease.

Dr. John H. Dunnington: I have under my care at the present time a woman, aged 73, who has this disease in an advanced stage. In the past year she has lost the vision in one eye almost completely. In the other eye vision has remained good, and there has been no change in the fields. There has been no inflammatory condition in either eye, only a gradual atrophy of the optic nerve of the right eye.

Dr. Irving Pardee (by invitation): It has been my good fortune to have seen quite a few cases of Paget's disease. As far as the skull changes in the vertex are concerned, this case is not advanced, but here the extensive process seems to be around the sphenoidal region at the base. I have never seen a case in which the optic nerve was involved. I have one case under observation at the moment, an unusual type, which is unilateral; only the left half of the skull is involved. That involvement is not only of the vertex, but also of the base of the skull and the sphenoid region. There is an associated paralysis of the third nerve and of the sixth nerve, an unusual observation in this disease. It is an interesting fact to me that this patient shows much more involvement of the frontal sinus and maxillary portion of the skull than is seen in most of these cases. The majority of cases show an enlargement of the skull, but not of the maxillary portion of the skull. In that way it is a separate process, and the opposite of what occurs in acromegaly, namely, an overgrowth of the maxillary portions and the mandible, while there is no change in the skull itself. We have puzzled for years over the etiology of these diseases, but I do not believe that we are any nearer the solution of this problem than we were years ago. It is interesting that they occur in later life. I have seen four or five cases in women at the menopause, but whether the conditions are associated with the menopause, I do not know.

Pulsating Traumatic Exophthalmos. Dr. Henry H. Tyson.

A man, aged 48, with traumatic pulsating exophthalmos due to an arteriovenous aneurysm in the cavernous sinus, caused by a fracture of the left frontal bone at the brow extending through the orbit to the optic foramen, was presented. A ligation of the left common carotid artery was performed in two stages during 1926, which relieved the proptosis and the head noises for about two years; then occasional head noises began to recur, varying in tone, intensity and frequency; also at this time paresis of the left sixth nerve and slight proptosis of

the left eye occurred. These signs and symptoms persisted and later increased. The exophthalmometer reading was 28 in the right eye and 32 in the left. Vision in both eyes was 20/30. The visual fields were contracted one-half, with enlarged blind spots, which were demonstrated well with Dr. Evans' minute test objects. Pupillary reactions were normal, except in the left eye, which is sluggish to light. There was complete paralysis of the left sixth nerve with marked injection of the vessels of the ocular conjunctiva. The patient had had frequent punctate retinal hemorrhages, and the optic disk in the right eye had blurred edges superiorly. Tension was normal. Blood pressure was 130 systolic and 90 diastolic. Physical examination and blood tests gave negative results. As ligation of the carotids on both sides is liable to induce destructive cerebral changes, and as the patient claimed that pressing over or on the upper part of the old cicatrix causes the head noises to disappear, I am inclined to advocate another ligation of the left common carotid artery close to the site of the former ligation.

DISCUSSION

DR. RAYMOND C. DODD: May I ask what ligature you used?

DR. HENRY H. TYSON: Dr. Peck performed the operation and used fascia lata.

DR. GUERNSEY FREY: Is it not rather uncommon to retain good vision after the ligation of the common carotid?

DR. ARNOLD KNAPP: What are you going to do with this case?

DR. HENRY H. TYSON: As pressure over the carotid stops the noise, I propose to do another ligation on the left common carotid.

DR. DANIEL B. KIRBY: It may be interesting to know that some of these aneurysms heal spontaneously. I followed a patient at the New York Eye and Ear Infirmary and Bellevue Hospital for two years. He had had a head injury twelve years previously and a unilateral nonpulsating exophthalmos; the fifth and sixth nerves on that side were involved. When he finally came to autopsy, we found an aneurysm of the intracranial portion of the internal carotid artery which had healed spontaneously by laminated clotting.

DR. RAYMOND C. DODD: I had a similar case of cavernous sinus aneurysm some years ago in which there was a spontaneous occurrence without trauma. The patient was a middle-aged woman who discovered the condition when she woke up one morning. Roentgenograms were made to see if we could find any etiology or factor producing it, but results were negative. In the interim, a delay of two or three days, she developed many hemorrhages over the retina and loss of vision, so that she could see nothing but hand movements. Dr. Eagleton and I ligated the common carotid with a strip of fascia lata. That was about five years ago. Vision returned to normal. I have seen the woman every year. When last examined, vision was still 20/15 in that eye with correction. There was no outward appearance of any unusual characteristics, and the internal appearance of the eye was normal.

DR. ERNEST F. KRUG: May I mention a case that has been under my observation for some time? About five years ago a man was accidentally shot while walking on the street. Two bullets went through

his neck from the tip of the left mastoid, one coming out in his nose and the other in his left cheek. Both were extracted. He had profuse hemorrhage from his nasopharynx and nose, slight protrusion of the left eye and scattered hemorrhages in the fundus, with a slightly blurred nerve head. An aneurysm developed, the bruit of which was so loud that one could hear it at a distance of a foot on that side. Hearing was completely lost in the left ear. The patient heard the bruit with his right ear, but he gradually became accustomed to it. Within twenty-four hours after the accident, his left common carotid artery was tied. The left eye gradually receded, and the fundus cleared up. When last seen, he had normal vision. His only unpleasant symptom was the bruit of the aneurysm. We had not been able to decide just where this aneurysm was located.

DR. HENRY H. TYSON: In reply to Dr. Frey, the vision in both eyes is almost normal as stated. My desire was for a discussion on the frequency of recurrence in these cases. I do not think that they recur often. Previously I presented two cases of arteriovenous aneurysm in the cavernous sinus. One was nontraumatic, occurring when a woman singer was trying for a high note; the condition disappeared in three months under rest, intermittent digital compression and internal administration of potassium iodide, with resulting good vision. The other case was one of trauma; the origin was fracture at the base of the skull. All signs and symptoms were relieved in this case by a ligation of the external carotid artery performed by Dr. Knapp, but there was subsequent optic atrophy in the eye on the same side.

TWO CASES OF TUMOR OF THE OPTIC NERVE. DR. THOMAS H. JOHNSON.

A girl, aged 13 years, was brought to the Neurological Institute because she was backward in learning and the right eye protruded. I saw her in consultation in the service of Dr. Timme. The family history was irrelevant. Birth had been normal. When 7 months of age, the patient had an attack of sore throat and had to have both ear-drums punctured. She was slow in standing alone, talking and learning to walk. When the patient was 2 years of age, the mother noticed that there was weakness in walking and that the left leg was shorter than the right. A diagnosis of disease of the hip joint was made, and the patient wore a plaster cast for fourteen months. At the age of 5, she was ill with scarlet fever for about five weeks. During the summer preceding her entrance into the Institute her mother noticed that the right eye was more prominent than the left. There was no history of trauma. Vision was 20/40+ in the right eye, and 20/50+ in the left. The right eye moved in all directions with a free excursion but on the tangent screen there was homonymous diplopia to the right and crossed diplopia to the left. Exophthalmometer reading showed 20 in the right eye and 16 in the left. Tension was 23 Schiötz in each eye. The right pupil was 5.5 mm. and the left 5 mm. in size. The pupillary reactions were normal. There was edema of the right nerve head. No bruit was heard. Except for a refractive error, the left eye was normal. The visual fields were normal except the right blind spot was enlarged. The patient was small for her age and was mentally retarded. On the Terman

revision of the Binet-Simon test this child measured 8 years and 1 month, with an intelligence test of .6. This placed her in the feeble-minded group among the morons. Her teeth were very bad. The tonsils had been removed. The blood chemistry and the blood count were normal. The Wassermann reactions of the blood and the spinal fluid were negative. Roentgen examination showed the calvarium to be rather thin. The skull tended slightly toward the turricephalic type. Convolutional digitations were rather well defined in the dependent portion of the skull. The suture lines appeared normal. The sella was quite small and was partly closed in. Its capacity was distinctly below the average for a skull of this size. The sphenoid cells were fairly extensive and clear. The ethmoids were apparently poorly developed, but not cloudy. The frontal cells were practically absent. The left antrum appeared smaller than the right, and it contained a rounded shadow of slightly increased density. The right antrum was clear. The thorax did not show any abnormal shadow in the superior mediastinum. The lung fields were comparatively clear. The pelvic bones were comparatively normal. The left hip, however, had been the seat of a pathologic change which resulted in a shortening of the neck and a lessening of the angle with the shaft. There was flattening of the epiphysis but these changes seemed to be the result of some process active in the past. It seemed rather improbable that there was any active pathology in this region at present. The changes were not suggestive of an infection but more of so-called Perthes' disease.

The right optic canal, as measured roentgenologically by Goalwin, was 3.5 by 3.9 mm., and the left 3.5 by 4.3 mm.

A married woman, aged 29, was first seen in March, 1928. The family history was negative; there was no history of trauma and there was nothing in the past history that would have any bearing on the present condition. In August, 1925, she noticed that the right eye seemed larger than the left. Since that time there had been a progressive increase in the size of the right eye. In March, 1928, the condition of the eye was much the same as it is today. The ophthalmometer reading was 25 mm. in the right eye and 18 mm. in the left. Both eyes moved with a free excursion in all directions. There was no diplopia. Measured by the Schiötz tonometer, the tension was 13 in the right eye and 15 in the left eye. The right field had a defect of the upper nasal quadrant extending to within 10 degrees of the fixation point. The blind spot was enlarged, and there was a papilledema of 5 diopters. A complete physical examination gave negative results. Blood chemistry and Wassermann tests of the blood gave negative results. An intradermal test with old tuberculin showed positive results, but no great importance was attached to this. Roentgen examination showed no destructive changes in the bones entering into the formation of the orbit. The optic foramen of each eye was normal. Roentgenograms of the chest were negative. When last examined, the vision was 20/200 in the right eye and 20/20 + in the left. The exophthalmometer reading was 28 in the right eye and 18 in the left. The proptosis in the right eye had increased 3 mm. in the last two years. The interior of the right eye showed edema of the nerve head. About one disk diameter below the nerve the retina could be seen with a +.50 diopter lens, the highest part of the nerve head with a +6 diopter lens, and the

retina about a disk diameter above the disk with a +5 diopter lens. My interpretation of this was that the tumor was pressing on the upper part of the posterior surface of the globe. Under dilation with homatropine, refraction with a +3.25 sphere, +1.00 cylinder axis 120 showed vision to be 20/200 in the right eye; in the left eye, with a +1.25 sphere, vision was 20/20.

The differential diagnosis was: The negative roentgenogram of the orbits ruled out meningocele, osteoma and mucocele extending from one of the accessory sinuses. Absence of bruit and pulsation were against aneurysm, and repeated negative Wassermann reactions against gumma. It was more difficult to eliminate a dermoid, an angioma and a tuberculous process, but it was believed that the following signs ruled them out: the painless and slow development of the exophthalmos, with proptosis tending to coincide with a line of the axis of the orbit; free movement of the globe in all directions; the absence of a destructive process; edema of the optic nerve head, reduced vision and in the woman a difference of 2 diopters in the hyperopia. It was hoped that these two patients would consent to a surgical removal of the tumors and that at a later date a complete report could be made of the pathologic observations.

A brief abstract of Byers' monograph on intradural optic nerve tumors completed the report.

DISCUSSION

Dr. Lewis Webb Crigler: I presented the older patient before the section about three years ago, with a diagnosis of unilateral exophthalmos with choked disk. At that time the eye was proptosed directly forward, 8 mm. There was free motility in all directions, except directly backward on pressure. The nerve head showed a swelling of 2 mm., as compared with the surrounding retina which appeared perfectly normal. The blind spot was slightly enlarged and the upper nasal field quadrant contracted. Vision was 20/70. A thorough search revealed no local or constitutional cause for the condition. A tentative diagnosis of myxofibroma of the optic nerve was made, and operation advised. The patient did not return.

Dr. Arnold Knapp: The interesting feature of these two cases is the edema of the nerve head. The cases of tumor of the optic nerve I have seen presented the picture of optic atrophy, as one would expect from a tumor involving the optic nerve itself. If these are cases of tumor of the optic nerve, as I think they are, they must be tumors of the sheath, circumscribing the optic nerve so as to produce pressure on the vein, causing papilledema. This is the only way that we can explain that the vision has remained good for all these years. An exploratory operation on the orbit should be done, and then the operator can be guided by the conditions found.

Dr. Sigmund Agatston: It struck me that in the woman the optic neuritis had the appearance of ordinary choked disk, caused by intracranial pressure rather than lateral pressure. I do not think that it is caused by intracranial pressure. Choked disk is usually caused by distention of the sheath of the optic nerve. As Dr. Knapp suggested, the tumor may arise from the nerve sheath, but it is only fluid sur-

rounding the nerve that would give such an appearance of the disk. If there were definite pressure on the vein, we would be more likely to get dilatation of the veins and hemorrhages. As I understand, this patient never had hemorrhages, so that there is something atypical about this tumor of the nerve.

DR. THOMAS JOHNSON: An attending physician in the neurological service at Bellevue made a diagnosis of intracranial tumor in this case. It is not unusual to see cases of papilledema without hemorrhage in the retina.

PARTIAL OPTIC ATROPHY. DR. WEBB W. WEEKS.

A man, aged 45, gave a history of blurred vision below and to the left of one month's duration. There was no history of colds in the head. Central vision was 20/15. The lower two thirds of the visual field was scotomatous. Ocular tension was normal and the media clear. The optic disk showed a sharp margin, with an atrophic area in the upper half. The optic vessels appeared normal. A roentgenogram of the teeth was negative. The tonsils had been removed. An examination of the blood gave negative results. A roentgenogram showed no fractures involving the orbit; there was a normal optic foramen but hazy sinus cells were seen on the left side. The sella was normal. Drainage of the sinuses showed no frank pus. For three months the condition has remained the same.

TOTAL ECTASIA OF THE SCLERA. DR. JAMES W. SMITH.

E. N., a man, aged 36, appeared for examination on Jan. 14, 1930. Examination of the right eye gave negative results; vision was 20/20. At the age of 6, the patient sustained a self-inflicted penetrating wound of the left cornea by a piece of wire. He was treated at the New York Eye and Ear Infirmary by Dr. Bull, and enucleation was advised. At the ages of 9 and 11, he was treated by Dr. Skeel for severe pain in the left eye. An iridectomy was performed for relief from discomfort, probably due to secondary glaucoma. During the twenty-five years preceding the present examination there had been no pain, no further increase in the size of the globe. Enucleation was repeatedly advised, but was refused on account of the cosmetic deformity.

Examination showed the left globe to be enlarged to about one and one-half times the normal diameter. Beneath the upper lid an exceedingly large anterior scleral ectasia was seen. The cornea was diffusely clouded by numerous infiltrates. Vision was nil. The entire sclera was markedly thin and dimpled readily on pressure. The extreme thinness of the sclera was demonstrated by the application of an ordinary ophthalmoscope bulb to the lower lid in a darkened room; the transillumination was so clear that the minute details of the ciliary ectasia superiorly were easily seen. The patient was presented before the section to demonstrate the great elasticity of the sclera in youth. The patient had agreed to enucleation during the following week.

STEREOSCOPIC PHOTOGRAPHY AS APPLIED TO THE EYE. DR. WENDELL L. HUGHES.

This paper appears in full on p. 585 of this issue.

DISCUSSION

DR. DANIEL B. KIRBY: I think that Dr. Hughes is to be congratulated on devising this ingenious apparatus and contributing so much to the advancement of the photography of the eye. I have been interested in the matter of photography of the crystalline lens to record the changes in cataract. The photography of the lens is difficult first because of the Purkinje images which act as reflections and obscure the picture, and, secondly, because of the natural efflorescence of the lens. When the incident beam passes through the lens, it obscures the details. In cortical cataract the early changes, vacuoles, lamellar separation and watersplitting of the sutures, are well seen by special methods of illumination with the microscope and ophthalmoscope, and the facility of this observation cannot be duplicated with the camera. The transparency of any medium is to be judged by its ability to transmit light. There is no camera that will take a photograph of the red reflex of the fundus or of the effect of lens opacities on the fundus reflex. Lastly, stereoscopic photographs are necessary to record the location and the depth of the opacity of the lens. I hope that Dr. Hughes will continue this work and will be able to overcome more of these difficulties.

DR. LAURANCE D. REDWAY: I think that the best contribution that Dr. Hughes has made, for which he cannot have too much credit, is the effective device of introducing the filter in the shutter and also the small hole in the shutter, so that it does not completely close. One thing that has held back the facility of photography has been the difficulty of focusing the light beam properly on the eye. This device solves that difficulty in a way which cannot be further simplified, and for which Dr. Hughes deserves a great deal of credit.

One other observation on photographic methods: I think by using them not only do we obtain a picture of the eye that may be useful in demonstrations of this sort or in illustrating articles, but we are also afforded a new tool for the investigation of the pathologic changes in the media; we are not distinctly limited in our investigations of the microchemical reactions of the human eye in the living subject. I think the study of photographic methods, the study of the spectra, the development of certain vital stains, and their reactions under differing conditions may throw a great deal of light on, and open up a vast new field of investigation of the functional characteristics of the living eye and the early pathologic changes which are now not available. It is that hope which I should like to hold out to all those men who may have seen the beginnings of photography as the eventual goal which does not as yet appear.

DR. THOMAS JOHNSON: I should like to ask if this method is applicable to photography of the interior of the eye, particularly the optic disk.

DR. WENDELL L. HUGHES: In regard to Dr. Kirby's remark on the lens, I have found, as he has, great difficulty in getting much detail in the lens. He is especially interested in senile cataract in its earliest stages. The early stages that he mentioned, vacuoles, watersplitting of the fissures and swelling of the lens fibers, I have not been able to demonstrate satisfactorily in a photograph. There is one picture in the series that shows several spots of a congenital nature in the lens; these

stand out brilliantly, but they are of an entirely different structure from the ordinary changes of a senile cataract.

Dr. Redway just gave us a glimpse of what he is interested in. It opens up an entire new field in relation to physiologic optics in which most of us are lacking. When we come to consider filters and vital staining and things along that line, which Dr. Redway has at his fingertips, it is a little bit over our heads. Dr. Redway has done a great deal of work on that.

In regard to Dr. Johnson's question, this is not applicable to anything behind the posterior part of the lens. Dr. Redway's color work in regard to the fundus I think is the coming field for fundus photography.

HERPES ZOSTER OPHTHALMICUS LUETICA. DR. IRVING H. PARDEE.

This paper will be published in full in a later issue.

DISCUSSION

DR. ERNEST F. KRUG: I wonder how many of us have had a Wassermann test done in our cases of herpes zoster. I must admit that I have not.

DR. RALPH I. LLOYD: The association of syphilis with herpes is, of course, a new idea. I wish to ask the members, when they have cases of severe herpes with terrific pain, to remember the use of heavy dosage of x-rays applied to the ganglion of the affected side; and if the tension of the eye is elevated at the same time, I think they will be agreeably surprised to see both relief from pain and reduction of tension. This idea came to me from a paper by Müller of Vienna. I have tried it in three different cases, with success in each instance, as far as the pain and the tension were concerned.

DR. WENDELL L. HUGHES: Have you had any experience with injections of alcohol into the gasserian ganglion in these painful types of trigeminal neuralgia around the eye, with or without definite herpes ophthalmicus?

DR. IRVING PARDEE: I can agree strongly with Dr. Lloyd that roentgen therapy for herpes zoster is most effective. Of course when there is a syphilitic etiology that can be attacked by antisyphilitic treatment, it is well worth giving the patient the benefit of a cure.

Injections of alcohol into the trigeminal nerve are effective only if the second and third branches are involved. If the first branch, the ophthalmic branch, is involved, the injection is not effective, but the operative procedure on the trigeminal nerve has almost approached the stage of being a minor operation. When you see patients at the Neurological Institute who have had an operation on the trigeminal nerve up and around the third day, walking about the ward in perfect comfort, you will appreciate that this is so. In the first place, it can be done under local anesthesia; in the second place, the most recent method of approach is not to sever the entire root, but to sever a portion of the nerve root. In that way it is possible to eliminate the pain, while one avoids the danger of trophic disturbances in the eye. Most of the pain fibers seem to run in the lower part of the nerve root. Cutting those

pain fibers in the lower part of the root leaves the upper portion of the root passing to the ophthalmic branch in good trophic control to carry on its necessary function in the eye. Postoperative trophic disturbances of the eye are now rarely seen. I also think that the mortality of the operation as it is now done is about 1 per cent, whereas it used to be very high. We are coming to assume the attitude that only those patients who refuse operation should be given an alcoholic injection, because the operation is so satisfactory and the alcoholic injection is a temporary procedure even at best. Sometimes the anesthesia will last for three, four or six months, but more often it is of only very short duration.

Book Reviews

De Oculis. Eorumque Egritudinibus et Curis. By Benevenutus Grassus of Jerusalem. Translated with notes and illustrations from the first printed edition, Ferrara, 1474 A.D., by Casey A. Wood, M.D., LL.D. Stanford University, California: Stanford University Press, 1929.

Although original copies of the "De Oculis" are to be found in all our large libraries, and Albertotti and others have given a wide choice of published Latin texts, a translation into English was a desideratum. Mediaeval Latin is not the Latin of our school books; it bristles with difficulties of nomenclature and derivation, of punctuation and of abbreviation. Time and training are necessary for an extensive perusal of its texts. A translation is essential to leisurely reading. Thanks to Dr. Wood, the "De Oculis" now lies open for easy study by his English-speaking colleagues.

The printing press was set up at Ferrara in 1471. Three years later what is known as the Ferrara edition of the "De Oculis" was struck off, and it is this edition, one of the longest, that was chosen for translation. After the elimination of repeated phrases and sentences and irrelevant admonitions, the translator sets forth his abridged text in chapters of varying lengths, with appropriate headings. Dr. Wood's aim was to render into pleasing English the ideas which Benevenutus strove to express, while preserving something of the form and phraseology of the fifteenth century text. I have read the translation many times and have compared it with the Latin text, and I think that Dr. Wood's effort has been eminently successful.

The translation occupies fifty-six of the 102 pages of an artistic and beautifully printed book that reflects the highest credit on the Stanford University Press from which it issues; and as the size of the printed page, the column, and the type of the translation approximate those of the Ferrara edition, the brevity of the original is apparent. The remainder of the book is given over to a consideration of germane topics. A section devoted to the predecessors and successors of the Ferrara edition is a critical review of the evidence in the case of the "Liber de Oculo" of Johannes de Peckham versus the "De Oculis" of Benevenutus in the matter of priority of printing. After full consideration, Dr. Wood finds that the date of the Benevenutus work is 1474, while that of the "Liber de Oculo" is 1475. He accordingly upholds the opinion of Dr. Fielding Garrison that the "De Oculis" is the earliest printed book on the eye.

The author's review of the life of Benevenutus is important because of the light that it throws on the period of the "De Oculis." I will quote his concise summary: "Where there is no historical certainty every student of world events is entitled to a guess about life dates, and mine in the case of our author is that he was a Hebrew born in Judea some time during the eleventh century, that he read and spoke at least Hebrew, Italian, Provencal, and Arabic, that he was largely educated at the medical school of Salerno, that he practiced both in the Near East and

in several South European cities, and that, having acquired a wide reputation as a skilful operator on cataract and as a specialist in other ocular ailments, he settled as scholar and practitioner in Montpellier. Here, welcomed as teacher and physician and free from persecution on account of his faith, he apparently taught his art and wrote a book that soon became and long continued to be a *vade mecum* among medical men the world over."

A catalog of the codices and prints of the "De Oculis," which includes titles previously overlooked, a bibliography and five enlightening illustrations, fascimile pages from the 1474 "Editio Princeps" and earlier Benevenutus manuscripts, round out this notable contribution.

In giving the means easily to know Benevenutus and his "De Oculis," Dr. Wood has conferred a boon on his English-speaking colleagues that will be widely appreciated. The value of Dr. Wood's contribution lies, however, not so much in its concrete accomplishment, important though this is, as in the interest it excites in the broad field of historic research. A study of the "De Oculis" leads inevitably to a study of medieval history, and against the background of medievalism the work of Benevenutus takes on a fuller meaning. It is seen that it preserved for the Europe of its time all the little classic learning concerning the eye that survived the dark ages; that it made small but definite contributions to the subject of which it treated, and that it represented the maximum possible achievement under the conditions then prevailing. Above all, the "De Oculis" stood for the will to carry on, for literacy in an age of ignorance.

> For Age, long Age!
> Nought else divides us from the fresh young days,
> Which men call ancient; seeing that we in turn
> Shall one day be Time's ancients, and inspire.

W. Gordon M. Byers.

Medical Research Council—Report of the Committee on the Physiology of Vision. VI. Some Experiments on Peripheral Vision. By Myer Salaman. Price, 2 shillings, 6 pence net. London: His Majesty's Stationery Office, 1929.

The author believes that the perception of moving objects is distinct from other visual perceptions. The object of the experiments was to discover whether, when a moving object is observed peripherally, a perception of change of position is ever observed without perception of movement. Reports were obtained from six persons. The observer sat, fixating a point on a wall facing him. At his left was a screen with a circular opening of 12 cm., behind which a strip of paper was made to pass by means of electrically driven drums, and on it, at 12 cm. intervals, were disks of black paper 1 cm. in diameter.

The motor started, the observer was asked to fixate on the wall an excentric angle of 100 degrees, to decrease the latter in steps of 5 degrees and to report observations.

Three observers reported changes of position of the spots at excentric angles at which no movement could be perceived. Most observers noted periods of disappearance of the spots. All noticed a falling off of the perception of movement toward the periphery. Light is thrown

on these phenomena by the recent work of Adrian and Matthews, who noted the electric response in the optic nerve of the eel, when the retina was stimulated. This correspondence between the properties of the visual process of the eel and the peripheral visual process in man is evidence that human peripheral vision preserves much of the apparatus of more primitive types of vision.

In a second series of experiments lights were used in a dark room. The so-called illusory perception of movement, produced by successive stimulation of adjacent retinal points, was studied in the peripheral field. The first object consisted of two pinhole lights horizontally disposed, the distance between which could be varied; the second, a pinhole light moving along a path. Both were used on a perimeter with a radius of 1 meter. Ten observers were employed. Five who had previous knowledge of the subject obtained some degree of illusory perception of movement; the others did not. All complained of the extreme vagueness of the impression.

The author also describes some preliminary experiments on the peripheral perception of form. Lines and simple geometrical figures were the test objects, and the apparatus used is described carefully. The results obtained by four observers are described. Two of these viewed the objects peripherally only, and the results, given in great detail, are interesting. There is much work to be done in this field.

JOHN B. LYNCH.

Directory of Ophthalmologic and Otolaryngologic Societies *

FOREIGN

Oxford Ophthalmological Congress

Master: Dr. A. B. Cridland, Salisbury House, Chapel Ash, Wolverhampton, England.
Secretary: Dr. C. G. Russ-Wood, 12 St. John's Hill, Shrewsbury, England.
Place: Oxford. Time: July 10-12, 1930.

Deutschen Ophthalmologischen Gesellschaft

Secretary: Prof. A. Wagenmann.
Place: Heidelberg. Time: June 12-14, 1930.

NATIONAL

American Medical Association, Section on Ophthalmology

Chairman: Dr. T. B. Holloway, 1819 Chestnut St., Philadelphia.
Secretary: Dr. William C. Finnoff, Imperial Bldg., Denver.
Place: Detroit. Time: June 23-27, 1930.

American Academy of Ophthalmology and Oto-Laryngology

President: Dr. William H. Wilder, 122 S. Michigan Ave., Chicago.
President-Elect: Dr. J. F. Barnhill, Miami Beach, Florida.
Executive Secretary: Dr. William P. Wherry, Medical Arts Bldg., Omaha.
Place: Chicago. Time: October, 1930.

American Ophthalmological Society

President: Dr. W. Campbell Posey, Radnor, Pa.
Secretary: Dr. Emory Hill, 501 E. Franklin St., Richmond, Va.
Place: Hot Springs, Va. Time: June 2-4, 1930.

SECTIONAL

New England Ophthalmological Society

President: Dr. S. Judd Beach, 704 Congress St., Portland, Maine.
Secretary-Treasurer: Dr. E. B. Dunphy, 520 Commonwealth Ave., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

Pacific Coast Oto-Ophthalmological Society

President: Dr. Arthur Jones, 105 N. 8th St., Boise, Idaho.
Secretary-Treasurer: Dr. Walter F. Hoffman, 817 Summit Ave., Seattle.
Place: Victoria, B. C. Time: September 4-6, 1930.

Puget Sound Academy of Ophthalmology and Oto-Laryngology

President: Dr. Walter F. Hoffman, 817 Summit Ave., Seattle.
Secretary-Treasurer: Dr. Francis A. Brugman, Medical-Dental Bldg., Seattle.
Time: 8 p. m., third Tuesday of each month, except July and August.

Southern Medical Association, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Fred Hasty, 119 Seventh Ave., Nashville, Tenn.
Secretary: Dr. Fletcher D. Woodward, Box 162, University, Va.
Place: Louisville, Ky. Time: November, 1930.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

STATE

Colorado Ophthalmological Society

President: A presiding officer is selected for each meeting alternately until all members have served.
Secretary: Dr. Donald H. O'Rourke, 217 Imperial Bldg., Denver.
Place: First floor, Metropolitan Building, Denver. Time: 7:30 p. m., third Saturday of the month, October to April, inclusive.

Eye, Ear, Nose and Throat Club of Georgia

President: Dr. George B. Smith, Rome.
Secretary-Treasurer: Dr. William O. Martin, Jr., 384 Peachtree St., Atlanta.

Indiana Academy of Ophthalmology and Oto-Laryngology

President: Dr. H. A. Van Osdol, 320 N. Meridian St., Indianapolis.
Secretary: Dr. Robert J. Masters, 23 E. Ohio St., Indianapolis.
Place: Indianapolis. Time: December 10 and 11, 1930.

Minnesota Academy of Ophthalmology and Oto-Laryngology

President: Dr. C. N. Spratt, 900 Nicollet Ave., Minneapolis.
Secretary-Treasurer: Dr. J. H. Morse, 80 S. 7th St., Minneapolis.

Montana Academy of Oto-Ophthalmology

President: Dr. Lee W. Smith, 125 W. Granite St., Butte.
Secretary: Dr. J. G. Parsons, Lewistown.

North Dakota Academy of Ophthalmology and Otolaryngology

President: Dr. George M. Constans, Bismarck.
Secretary-Treasurer: Dr. F. L. Wicks, Valley City.

Oregon Academy of Ophthalmology and Oto-Laryngology

President: Dr. J. W. McCollom, 346½ Washington St., Portland.
Secretary-Treasurer: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

Rhode Island Ophthalmological and Otological Society

President: Dr. J. E. Raia, 276 Broadway, Providence, R. I.
Secretary: Dr. F. W. Dimmitt, 195 Thayer St., Providence, R. I.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

Utah Ophthalmological Society

President: Dr. F. M. McHugh, 17 Exchange Pl., Salt Lake City.
Secretary-Treasurer: Dr. H. Leroy Smith, Ezra Thompson Bldg., Salt Lake City.

Virginia Society of Oto-Laryngology and Ophthalmology

President: Dr. Fletcher D. Woodward, Box 162, University.
Secretary-Treasurer: Dr. Henry Grant Preston, Harrisonburg.

LOCAL

Academy of Medicine of Northern New Jersey, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Frederick J. Wort, 1080 Broad St., Newark.
Secretary: Dr. Earl LeRoy Wood, 31 Lincoln Park, Newark.
Place: 91 Lincoln Park South. Time: 8:45 p. m., second Monday of each month.

Academy of Medicine of Toronto, Section of Ophthalmology

Chairman: Dr. Louis Joseph Sebert, 86 Bloor St., W., Toronto.
Secretary: Dr. Alexander E. MacDonald, 151 Bloor St., W., Toronto.
Time: First Monday of winter months.

ATLANTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Leon E. Brawner, 157 Forrest Ave., Atlanta, Ga.
Secretary: Dr. William O. Martin, Jr., 384 Peachtree St., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St. N. E. Time: 8 p. m., last Thursday of each month.

BALTIMORE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Leo J. Goldbach, 1012 N. Charles St., Baltimore.
Secretary: Dr. Angus Lloyd MacLean, Johns Hopkins Hospital, Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

BROOKLYN OPHTHALMOLOGICAL SOCIETY

President: Dr. Robert M. Rogers, 680 St. John's Pl., Brooklyn.
Secretary: Dr. William F. C. Steinbugler, 515 Ocean Ave., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday of February, April, May, October and December.

BUFFALO OPHTHALMOLOGIC CLUB

President: Dr. E. E. Blaauw, 190 Ashland Ave., Buffalo.
Secretary-Treasurer: Dr. A. F. Luhr, Medical Arts Bldg., Buffalo.

CHICAGO OPHTHALMOLOGICAL SOCIETY

President: Dr. Charles G. Darling, 30 N. Michigan Ave., Chicago.
Secretary: Dr. Richard C. Gamble, 30 N. Michigan Ave., Chicago.
Place: Medical and Dental Arts Club. Time: Third Monday from October to May.

CLEVELAND ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

Chairman: Dr. M. Paul Motto, Rose Bldg., Cleveland.
Secretary: Dr. A. D. Ruedemans, Euclid and E. 93d St., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

CLEVELAND OPHTHALMOLOGICAL CLUB

Chairman: Dr. W. E. Bruner, Guardian Bldg., Cleveland.
Secretary: Dr. M. P. Motto, Rose Bldg., Cleveland.

COLLEGE OF PHYSICIANS, PHILADELPHIA, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Maxwell Langdon, 1530 Locust St., Philadelphia.
Clerk: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

COLUMBUS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

Chairman: Dr. John E. Brown, 370 E. Town St., Columbus, Ohio.
Secretary-Treasurer: Dr. Albert D. Frost, 681 E. Broad St., Columbus, Ohio.
Time: First Monday of each month.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. D. L. Bettison, Medical Arts Bldg., Dallas, Texas.
Executive Secretary: Dr. W. Mood Knowles, Medical Arts Bldg., Dallas, Texas.
Recording Secretary: Dr. L. A. Nelson, 4105 Live Oak, Dallas, Texas.
Place: Dallas Athletic Club. Time: 6 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

DETROIT OPHTHALMOLOGICAL CLUB

Chairman: Members rotate alphabetically.
Secretary: Dr. Parker Heath, David Whitney Bldg., Detroit.
Time: 6:30 p. m., first Wednesday of each month.

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. John P. O'Keefe, 251 State St., Albany.
Secretary-Treasurer: Dr. Arthur F. Holding, 142 Washington Ave., Albany.
Time: Third Wednesday of October, November, March, April, May and June.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. W. R. Thompson, 510 Main St., Fort Worth, Texas.
Secretary-Treasurer: Dr. R. H. Needham, Fort Worth National Bank Bldg., Fort Worth, Texas.
Place: University Club. Time: 6:30 p. m., first Friday of each month except July and August.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. Claude C. Cody, 1306 Walker Ave., Houston, Texas.
Secretary: Dr. J. M. Robison, Eye, Ear, Nose and Throat Hospital, Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., first Thursday of each month from September to June.

Indianapolis Ophthalmological and Oto-Laryngological Society

President: Dr. Robert J. Masters, Hume-Mansur Bldg., Indianapolis.
Secretary: Dr. Kenneth L. Craft, Hume-Mansur Bldg., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

Kansas City Society of Ophthalmology and Otolaryngology

President: Dr. L. R. Forgrave, Logan Bldg., St. Joseph, Mo.
Secretary: Dr. A. E. Eubank, Argyle Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

President: Dr. W. B. Wright, Jr., 1209 Pine Ave., Long Beach, Calif.
Secretary-Treasurer: Dr. M. A. Schurter, 114 E. 7th St., Long Beach, Calif.
Place: Pacific Coast Club. Time: Last Wednesday of each month from September to June.

Los Angeles County Medical Society, Section on Ophthalmology and Otolaryngology

President: Dr. A. Ray Irvine, 727 W. 7th St., Los Angeles.
Secretary-Treasurer: Dr. J. Frank Friesen, 727 W. 7th St., Los Angeles.
Place: California Lutheran Hospital, Assembly Room. Time: 8 p. m., first Monday of each month.

Medical Society of the District of Columbia, Section of Ophthalmology and Oto-Laryngology

Chairman: Dr. Louis S. Greene, 1710 Rhode Island Ave. N. W., Washington.
Secretary: Dr. E. R. Gookin, Medical Science Bldg., Washington.
Place: 1718 M St. N. W. Time: 8 p. m., third Friday of each month.

Memphis Society of Ophthalmology and Oto-Laryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. R. O. Rychener, Exchange Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

Milwaukee Oto-Ophthalmic Society

President: Dr. Edward R. Ryan, 425 E. Water St., Milwaukee.
Secretary-Treasurer: Dr. O. P. Schoofs, 120 E. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6:30 p. m., third Tuesday of each month.

NASHVILLE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. R. J. Warner, 706 Church St., Nashville, Tenn.
Secretary-Treasurer: Dr. W. W. Wilkerson, Jr., Bennie-Dillon Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month.

NEW ORLEANS OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. W. A. Wagner, Pere Marquette Bldg., New Orleans.
Secretary-Treasurer: Dr. M. F. Meyer, Canal Bank Bldg., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: Third Thursday of each month from October to June.

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Chairman: Dr. E. F. Krug, 12 W. 44th St., New York.
Secretary: Dr. William F. C. Steinbugler, 815 Park Ave., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

OMAHA AND COUNCIL BLUFFS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. Charles M. Swab, Medical Arts Bldg., Omaha.
Secretary-Treasurer: Dr. J. C. Davis, 1615 Howard St., Omaha.
Place: Medical Tea Room. Time: 5:15 and 7:00 p. m., third Wednesday of each month from October to May.

PITTSBURGH OPHTHALMOLOGICAL SOCIETY

President: Dr. Edward B. Heckel, Jenkins Arcade Bldg., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

PITTSBURGH SLIT-LAMP SOCIETY

President: Dr. W. W. Blair, 121 University Pl., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: 121 University Pl. Time: Second Monday evening of every month, except June, July, August and September.

ROCHESTER EYE, EAR, NOSE AND THROAT SOCIETY

Chairman: Dr. A. Vincent Walker, 833 South Ave., Rochester, N. Y.
Secretary-Treasurer: Dr. John F. Gipner, 53 Fitzhugh St., Rochester, N. Y.
Place: Rochester Medical Assn., 113 Prince St. Time: 8 p. m., third Monday of each month from October to May.

ST. LOUIS OPHTHALMIC SOCIETY

President: Dr. Charles W. Tooker, 308 N. 6th St., St. Louis.
Secretary: Dr. Carl T. Eber, 1006 Carleton Bldg., St. Louis.
Place: St. Louis or Washington University Cafeteria.
Time: 6:30 p. m., fourth Friday of each month from October to April, inclusive.

SAN ANTONIO OPHTHALMOLO-OTO-LARYNGOLOGICAL SOCIETY

President: Dr. F. W. Sorrell, Gibbs Bldg., San Antonio, Texas.
Secretary-Treasurer: Dr. A. F. Clark, 728 Main Ave., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

SPOKANE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. A. N. Codd, 421 W. Riverside Ave., Spokane, Wash.
Secretary: Dr. Marc Anthony, Old National Bank Bldg., Spokane, Wash.

SYRACUSE EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. George H. Rockwell, Physicians Bldg., Syracuse, N. Y.
Secretary-Treasurer: Dr. A. Harry Rubenstein, Medical Arts Bldg., Syracuse, N. Y.
Place: University Club. Time: Second Friday of each month except June, July and August.